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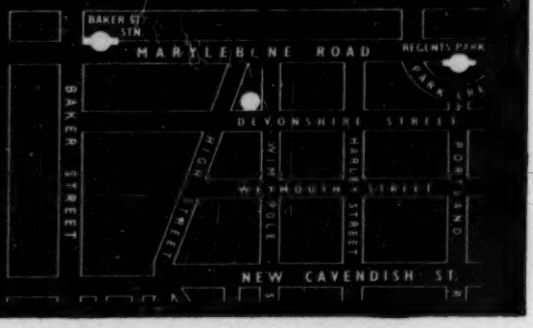

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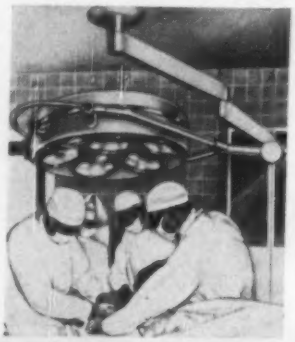

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BRITISH JOURNAL OF ANAESTHESIA

VOL. XXXII, No. 1 JANUARY 1960

EDITORIAL

DRUG ADDICTION

THERE will be general agreement with the first part of the interim report of the Inter-departmental Committee on Drug Addiction.* This committee, presided over by Sir Russell Brain, appears to be doing an excellent job of work, and its recommendation that in general "any drug or pharmaceutical preparation which has an action on the central nervous system and is liable to produce physical or psychological deterioration should be confined to supply on prescription" seems long overdue. There will be widespread hope that if implemented with expedition this may prevent the people of this country becoming "tranquillizer addicts". It is to be hoped that this recommendation will apply to those numerous drugs available for weight reduction, the excessive and unwise use of which may be disastrous. The report further recommends that "an independent expert body" should be responsible for advising which substances should be so controlled. This is again long overdue.

Anaesthetists are known to have given evidence before this commission and their testimony is reflected in the section of the report devoted to addiction to anaesthetic gases. While it was impossible to determine accurately the incidence of addiction of this kind, it appeared that in the past eleven years under twenty cases have come to notice

It should be remembered that there are some 1,500 doctors exclusively practising anaesthesia, excluding a number of general practitioners, resident medical staff, etc. Furthermore, all these cases "would seem to have been of essentially abnormal personality". The report concludes that the incidence of this irregularity has been very small indeed. The committee accepted the need for anaesthetists occasionally to sample by sniffing the mixture they are delivering in these words: "It was represented to us that with the apparatus at present in use, the preliminary sniffing of the gases immediately before administering them to the patient is a recognized and indispensable precaution. Neglect of this measure in the first place might be tantamount to professional negligence. Furthermore, of the great majority of anaesthetists, such a practice offers no encouragement to addiction." With these views we are in complete agreement.

The committee does not make recommendations as to how cases of irregularity in this matter are to be dealt with. There are discussions proceeding at the moment with the Ministry with the object of arriving at a satisfactory procedure. We would only point out that this is a matter which must be left to the medical profession and there is little doubt that with encouragement the medical staffs of hospitals are well able to deal with cases as they arrive. Failure to do so in the past has been uniquely exceptional and should not demand the establishment of any special committee of individuals to supervise and pass judgement on their colleagues. Indeed, it might be that with the passage of time the individuals on these committees might be the ones requiring advice or admonition.

* *Drug Addiction.* Interim report of the Inter-departmental Committee. H.M. Stationery Office, price 6*d*.

Brit. J. Anaesth. (1960), 32, 2

THE INFLUENCE OF MORPHINE AND PETHIDINE IN COMBINATION WITH LEVALLORPHAN ON BILIARY DUCT PRESSURE AFTER CHOLECYSTECTOMY

BY

K. KJELLGREN

The Department of Surgery, Central Hospital, Gelfe, Sweden

IT has long been known that morphine causes contraction of the sphincter of Oddi (Kitakoji, 1930) and thereby raises the pressure in the biliary ducts. Gaensler, McGowan and Henderson (1948) demonstrated that pethidine had a similar action, although the rise in pressure in the ducts was somewhat less. The opiate antidote (-)-3-oxy-N-allyl-morphinan (levallorphan) is in several respects antagonistic to morphine and pethidine (Fromherz and Pellmont, 1953; Lendle, 1953; Malorny 1955). One may therefore wonder if this antagonism influences the action of morphine and pethidine on the sphincter of Oddi. Although Lipchik (1958) studied the effect of levallorphan on morphine-induced spasm in this sphincter, the smallness of his clinical material and the absence of controls precluded reliable inferences.

In the present study the pressure in the biliary ducts was measured in cholecystectomized patients after administration of morphine or pethidine alone and in combination with levallorphan.

MATERIAL AND METHODS

It is generally agreed that the sphincter of Oddi seems always to react to morphine by contraction. In hitherto unpublished experiments, however, I found that this reaction may be absent if the sphincter mechanism is disturbed by calculi in the common bile duct, inflammatory changes there or in the pancreas, or manipulations in the vicinity of the sphincter during operation. In consideration of these findings, the present study was limited to patients with uncomplicated cholecystolithiasis.

A series of 20 patients was investigated after the injection of morphine and morphine levallorphan. A further 10 patients were given pethidine and pethidine-levallorphan (Pethilorfan).

Cholangiography was routinely performed during cholecystectomy. At the end of the operation a cannula was inserted into the common bile duct through the stump of the cystic duct or through a choledochotomy. Pressure readings were taken 3 to 5 days after the operation. The biliary duct pressure was then conducted via the drain either to a simple indicator tube, where it was read against a scale graduated in centimetres, or to an electromanometer, where the tension changes caused by the fluctuations in pressure were taken up by a potentiometer, amplified and traced on paper by a recorder. The accuracy of registration in both procedures was 0.5 cm of water. The patients were supine during the pressure measurements. As zero point the confluence of the cystic duct and the hepatic duct was selected. The level of this was marked on the skin of the right flank during cholecystectomy.

Only resting pressures were registered. For present purposes no interest was attached to pressures during perfusion of fluid and subsequent residual pressures.

Each patient was studied twice, viz., after morphine and morphine-levallorphan or after pethidine and pethidine-levallorphan. As a rule the observations were made on two successive days. To eliminate the time factor as far as possible, half of the patients in the respective groups received morphine or pethidine alone on the first day, while the other half were given the same drug combined with levallorphan, and on the second day this arrangement was reversed.

The dose of morphine was always 10 mg. When morphine and levallorphan were given together, the dose of the latter was as follows:

0.5	mg	levallorphan	in	3 cases
1.0	„	„	„	3 „
1.5	„	„	„	2 „
2.0	„	„	„	12 „

The doses in the "pethidine group" were 100 mg of pethidine with or without 1.25 mg of levallorphan.

The drugs were given by subcutaneous injection. Paired drugs were injected simultaneously.

RESULTS

Statistical treatment of the results would have been desirable but, because of the many individual variables that influence such results, this was considered to be scarcely warrantable. The wide variations in initial pressure (5.0–15.5 cm of water) in biliary flow and in intestinal function suffice as examples. One must therefore have recourse to comparison of observations in individual patients and means of these values.

All the diagrams are based on the increase of biliary duct pressure following administration of the various drugs. The pressure before the injection thus is stated as zero in all cases.

In figure 1 the effect of 10 mg of morphine may be studied. All 20 patients reacted with pressure elevation, which 60 minutes later had subsided in only 1 patient. In 17 patients the pressure was still elevated 90 minutes after the injection. The highest value on the graph of means was 5.6 cm of water.

Figure 2 shows the pressure measurements following injection of 10 mg of morphine with levallorphan according to the stated distribution of dosage. In 2 cases no pressure elevation resulted. In 8 of the other patients the pressure had returned to initial levels after 60 minutes. Ninety minutes after the injection only 6 of the patients still had elevated biliary duct pressure. The highest mean value was 4.7 cm of water.

Thus it seemed that the addition of levallorphan shortened the duration of the pressure rise produced by morphine, and that this rise was generally less after the combined drugs.

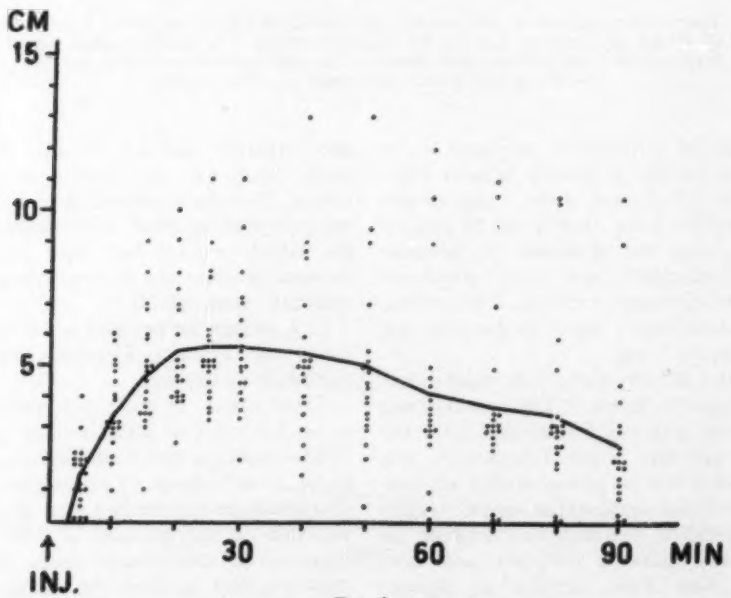

FIG. 1

The pressure increase in the common bile duct after the subcutaneous injection of 10 mg of morphine. The dots indicate individual readings and the graph denotes the mean of these readings.

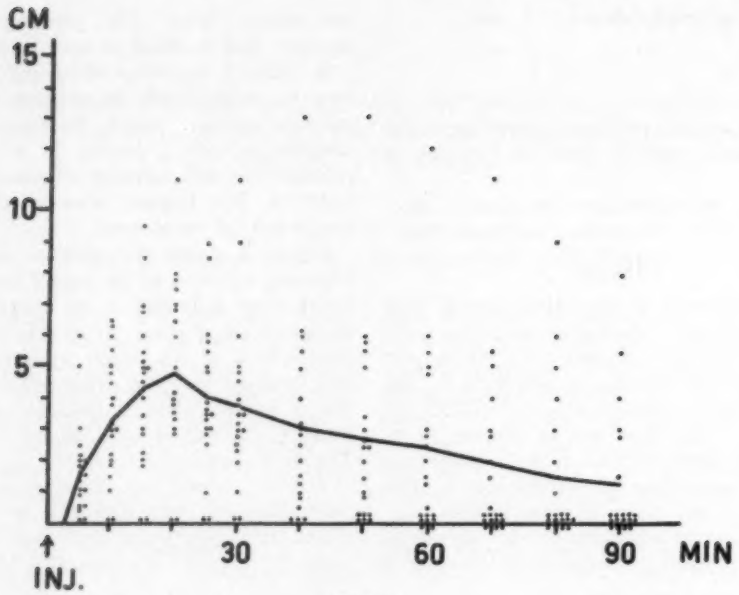

FIG. 2

The pressure increase in the common bile duct after the subcutaneous injection of 10 mg of morphine and 0.5–2.0 mg levallorphan. The measurements were made on the same patients as in figure 1. The dots indicate individual readings and the graph denotes the mean of these readings.

The amount of levallorphan appeared to be less important. Of the 2 patients without pressure elevation, 1 had been given 1 mg of this drug, and the other 2 mg. In 4 of the 20 patients no antidote action was observed; the pressure curves after morphine and after morphine-levallorphan being almost identical. Three of the 4 patients had received 2 mg of levallorphan and the fourth patient 1 mg.

The influence of pethidine on the biliary duct pressure is shown in figure 3. The elevation was less pronounced and was briefer than after the injection of morphine alone. Comparison with figure 4, which shows the pressures after administration of pethidine-levallorphan gives roughly the same impression as comparison between the pressure curves following morphine and morphine-levallorphan. Thus, elevation of pressure occurred in all of the 10 patients after pethidine alone and in 8 patients after pethidine-levallorphan The highest mean readings were 3.3 cm of water after pethidine and 2.2 cm after the combined drugs. Here, too, the duration of the rise was shorter after the combined drug injection. Sixty minutes after injection of pethidine-levallorphan the initial reading had been regained in 9 patients, whereas the corresponding figure after pethidine alone was 4.

In 1 patient the pressure curve after pethidine alone was virtually identical with that after combined medication.

Levallorphan in these experiments gave rise to no subjective or objective side effects.

The results of the investigations may be summarized as follows. Levallorphan seemed to counteract the contractile action of morphine and pethidine on the sphincter of Oddi. The rise in pressure in the biliary ducts that followed administration of these two drugs usually was reduced and shortened by simultaneous administration of levallorphan. In a few cases no pressure rise occurred when levallorphan was given.

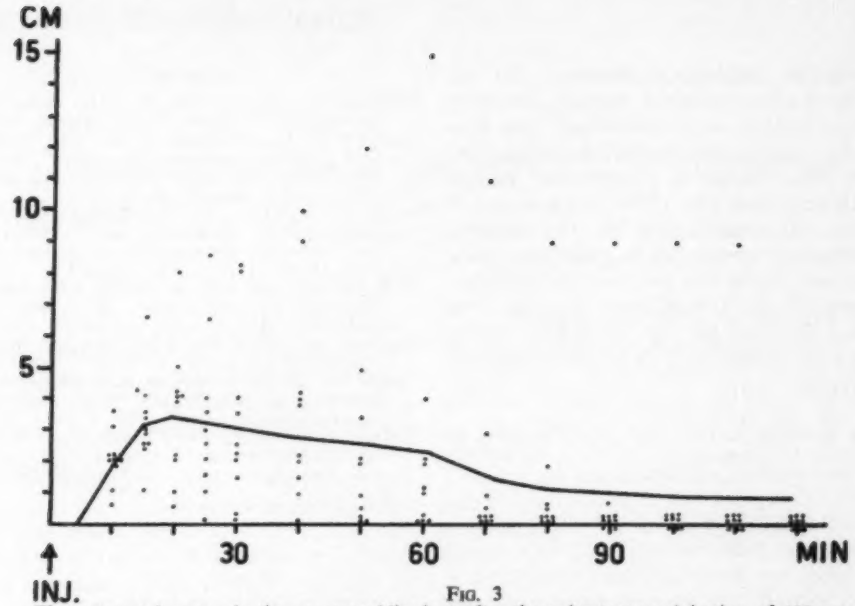

FIG. 3
The pressure increase in the common bile duct after the subcutaneous injection of 100 mg of pethidine. The dots indicate individual readings and the graph denotes the mean of these readings.

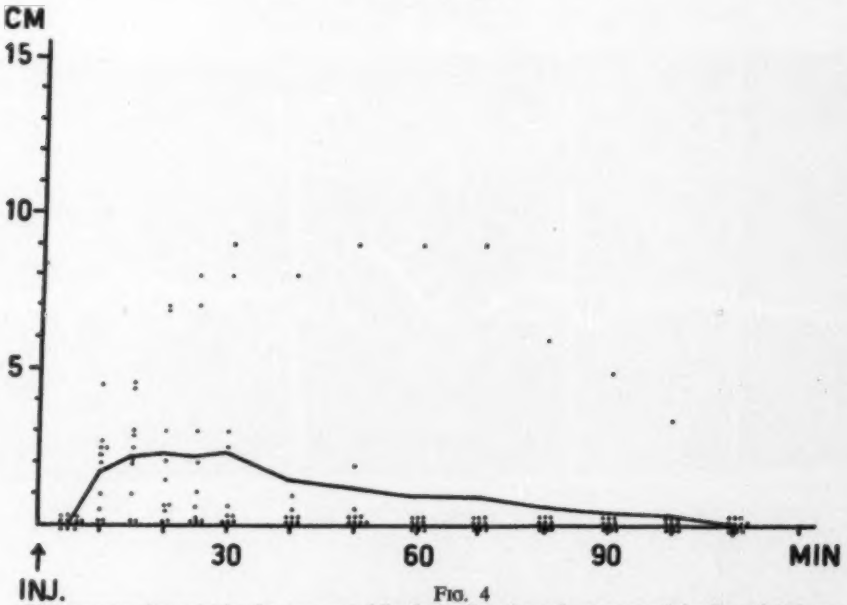

FIG. 4
The pressure increase in the common bile duct after the subcutaneous injection of 100 mg of pethidine and 1.25 mg of levallorphan. The measurements were made on the same patients as in figure 3. The dots indicate individual readings and the graph denotes the mean of these readings.

As regards pethidine-levallorphan, the reported results have received practical testing in a series of patients with cholelithiasis who were given this combination (pethidine-levallorphan) together with atropine as pre-operative medication (Kjellgren and Löf, 1959). Comparisons of the effect of premedication on the operative cholangiograms were made between these patients and others who had received morphine with hyoscine or a barbiturate together with atropine. The frequency of satisfactory cholangiograms was highest after pethidine-levallorphan and atropine.

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blood pressure began to fall within 4 seconds of

The vagolytic action of gallamine demon-

TRANSIENT HYPOTENSION IN THE CAT INDUCED BY GALLAMINE*

BY

J. P. PAYNE

Department of Anaesthesia, Postgraduate Medical School, London, W.12

WHILE investigating certain aspects of the neuromuscular blocking properties of gallamine in cats it was observed that in some animals the intravenous injection of small quantities of the drug was followed by a transient hypotension. As any activity displayed by a drug in the experimental animal may also reveal itself in clinical practice (Paton, 1959) it seemed desirable to examine this phenomenon more closely.

In a series of twenty cats hypotension occurred on four occasions. Characteristically the fall in blood pressure was constant for each animal (fig. 1) although the effect varied from cat to cat. The smallest change was a fall of 20 mm Hg, while the largest was over 100 mm. The remaining two dropped by 30 and 50 mm Hg respectively.

In one cat anaesthetized with chloralose it was possible to investigate the hypotensive response in some detail (fig. 1). In this animal a dose of gallamine (0.25 mg/kg) too small to produce any neuromuscular blocking effects was sufficient to lower the blood pressure by 50 mm Hg. On analyzing this response it was found that the

*Work done in the Department of Pharmacology, Royal College of Surgeons of England.

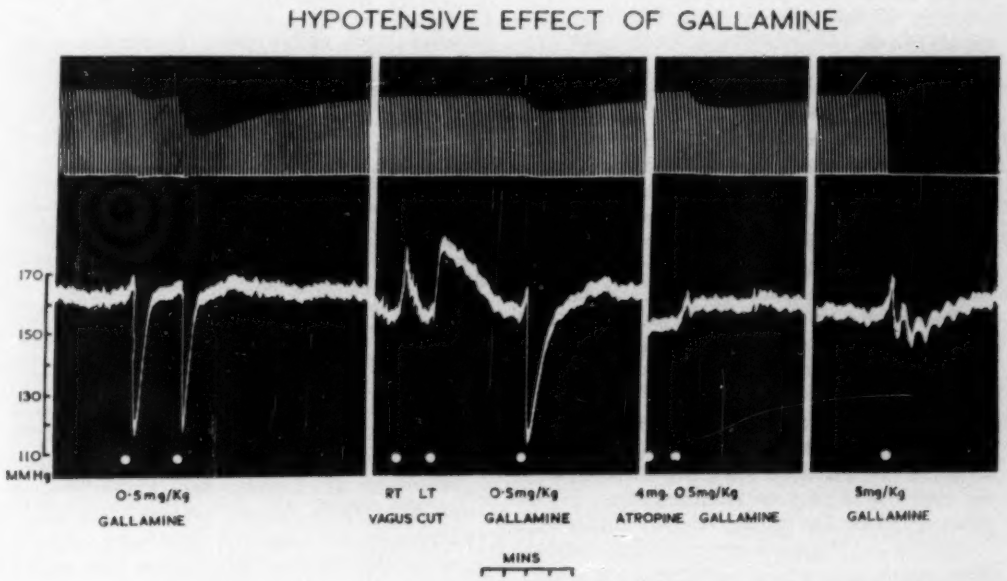

FIG. 1

Cat, male, 3.4 kg. chloralose. Ant. tibialis contractions in response to indirect stimulation. Blood pressure. Hypotensive effect of gallamine uninfluenced by bilateral vagal section but antagonized by atropine. No effect on neuromuscular blocking action.

blood pressure began to fall within 4 seconds of the injection of gallamine into the external jugular vein and the limit of the response was reached in a further 8 seconds. Recovery began 15 seconds later and was usually complete within 45 seconds of the time of the injection.

Doubling the dose and increasing it tenfold did not enhance the fall in blood pressure. Repeated doses (6) at 2-minute intervals did not modify the response nor did the provision of an interval of 60 minutes between injections. The division of both vagus nerves had little effect on the extent of the blood pressure fall although from figure 1 it is obvious that the recovery time has been prolonged. But when the injection of gallamine was preceded by the administration of intravenous atropine (4 mg) 1 minute beforehand, the hypotensive response was abolished. That this was a true atropine antagonism was shown by the fact that after sufficient time had been allowed for the effects of atropine to wear off gallamine again produced hypotension.

It is perhaps worth noting that despite the marked fall in blood pressure, the pulse rate was relatively slightly altered from 130 beats per minute to 145.

DISCUSSION

When hypotension follows the intravenous injection of a drug several possibilities need to be considered. A central action is not uncommon with many drugs and the possibility of vagal effects should not be ignored. Ganglionic block may be a side effect of certain relaxant drugs and histamine release is common to all of them. Finally, in this group the possibility of an anticholinesterase activity should be examined.

Except under markedly artificial conditions, the possibility of a central action by gallamine is too remote to be considered. The drug is a highly ionized compound containing three quaternary ammonium groups; such substances do not cross the blood-brain barrier and cannot act centrally when given intravenously.

The vagolytic action of gallamine demonstrated by Riker and Wescoe (1951) makes it unlikely that the fall in blood pressure is due to vagal stimulation, and the occurrence of hypotension when gallamine was injected after the division of both vagus nerves confirms this conclusion.

Riker and Wescoe further investigated the action of gallamine on ganglionic transmission and showed that even with doses as high as 10 mg/kg injected intra-arterially into cats there was no depression of transmission across ganglia. They concluded that gallamine cannot be regarded as a ganglionic blocking drug.

While histamine release must always be remembered when considering relaxant drugs, the absence of the typical response to histamine as described by Paton (1959) seems to exclude this possibility in the experiments under discussion.

According to Foldes (1957) all relaxant drugs show some anticholinesterase activity and the fact that the hypotension in this case was antagonized by atropine supports this view. Moreover, the pattern of response is closely similar to that obtained when acetylcholine is injected intravenously into cats in large doses (Payne—unpublished observations).

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ENDOTRACHEAL ASPIRATION AND OXYGENATION IN RESUSCITATION OF THE NEWBORN

BY

R. J. HAMER HODGES, M. E. TUNSTALL, R. F., KNIGHT,* AND E. J. WILSON

The Obstetric Unit, St. Mary's Hospital, Portsmouth

THE indications for active intervention in neonatal resuscitation vary from hospital to hospital. There is little agreement as to whether the clinical control should be the immediate responsibility of the obstetrician, the paediatrician or the anaesthetist (Schmidt, McLandress and Cruickshank, 1956), and which of a large variety of methods should be used (Abramson, 1956).

Most physicians now agree that the administration of oxygen takes precedence over the administration of drugs, but there is no general agreement concerning the route of administration. The shortcomings associated with the administration of oxygen by mask are now appreciated and recently the effectiveness of intragastric oxygen, first advocated by Akèrrén and Fürstenburg (1950) and Waller and Morris (1953), has been questioned by Holme and Payne (1955), James et al. (1959) and Barrie (1959). Osborn (1958) has further shown that indwelling oesophageal catheters are not without their dangers, and rupture of the stomach due to overdistension has followed this method of resuscitation (Hodges, 1956).

It has been alleged that endotracheal intubation may lead to shock and trauma, so increasing the infant mortality rate (Bloxsom, 1951). Some therefore regard this procedure as contra-indicated (*Lancet*, 1951); especially in small infants (Lord, Powell and Roberts, 1953). Laryngeal ulceration has been cited as a complication, and even among those who advocate it, intubation is not always regarded as a primary measure (Peddie, 1957).

In this unit during operative obstetric deliveries when an anaesthetist is present the responsibility for neonatal resuscitation is, in the first instance, delegated to him.

In anaesthesia endotracheal suction and the administration of oxygen by intermittent inflation is an accepted form of therapy for obstructed, failing, or absent respiratory effort. We follow these principles which are applied to neonatal resuscitation also by others (Roberts, 1949; Kromm, 1955; Rees, 1958).

This paper analyzes the results obtained in 137 infants in whom endotracheal resuscitative measures were undertaken. Conclusions are drawn as to the difficulties, dangers and the effectiveness of the technique.

MATERIAL AND METHOD

All the patients were in-patients in the obstetric unit and formed a series of 754 operative procedures from an overall 6,574 patients delivered.

All the anaesthetics were administered by members of the anaesthetic department, and consisted of a sleep dose of thiopentone followed by suxamethonium, nitrous oxide and oxygen with endotracheal intubation. This method has been described, found to have advantages over the more traditional methods, and further to be associated with little or no neonatal depression referable to anaesthesia (Hodges et al., 1959, 1960).

After delivery the infant was placed on a special neonatal trolley near the head of the operating table. The infant lay on a padded rest which could be tilted as desired. Resuscitative equipment was available on this trolley.

Immediately after delivery, if the clinical observations suggested that aspiration of liquor or regurgitated gastric contents had occurred, endotracheal suction was undertaken.

After the establishment of a clear airway, with or without endotracheal suction, the indications for endotracheal oxygen and intermittent positive pressure inflation were signs of anoxia associated

*Present Appointment. Captain, R.A.M.C.

with apnoea, subsequently cyanosis, or failure to establish regular spontaneous respirations within 2 to 3 minutes. Each case was judged individually according to these criteria. Bradycardia and cardiac irregularities were regarded as indicative of anoxia. Intubation was carried out using size 00 Magill tube, or a Foregger shouldered tube. Oxygen was administered by manual compression of an open-ended bag on a T-piece circuit with manometric control. Pressures of between 30 and 40 cm H_2O were obtained, but were maintained for the shortest possible period. The movement of the chest was carefully observed. Suction was carried out as rapidly as possible, and was continued intermittently for short periods only. Between these periods of suction the infant was allowed to inhale oxygen, or in the absence of spontaneous respirations intermittent positive pressure inflation was continued. The infants were extubated as soon as the airway was cleared and regular spontaneous respirations were established.

All premature infants and those in whom the response to resuscitative measures was considered unsatisfactory, were transferred to the paediatric department.

The state of the infant at birth and the resuscitative measures applied were recorded on the anaesthetic-obstetric punch card previously described (Hodges, 1959). These observations together with the subsequent obstetric, paediatric and, when necessary, pathological records form the basis of this paper.

All stillbirths and neonatal deaths were classified at special monthly obstetric meetings held for this purpose by the obstetric department, and attended by representatives from the departments of paediatrics, anaesthesia, and pathology.

RESULTS

The group of 137 infants considered here represented 18 per cent of the operative deliveries, the incidence of intubation following Caesarean section being the highest. In 74 infants (54 per cent) pre-operative foetal distress in utero had been diagnosed. Six infants were delivered pulseless and apnoeic, being apparently stillborn. Resuscitative measures restored regular pulse and respirations in one patient but with short-lived success. Subsequently 11 other neonatal deaths took place.

Thirty-three per cent of the infants were premature according to their dates, and 15 per cent weighed less than 2,500 g. The majority of the group of premature infants was delivered by Caesarean section.

Most of the infants were intubated because of primary apnoea (or because they had only gasping respiratory efforts), subsequent clinical hypoxia, or failure to establish regular respiratory effort within 2 minutes of birth. Seventeen per cent of the infants breathed regularly at birth, but either relapsed later and had only a weak respiratory effort associated with hypoxia, or aspirated mucus and liquor and became obstructed or depressed. This was regarded as secondary respiratory depression. Twenty per cent of the infants suffered primarily from obstruction and were intubated for the purpose of aspiration (table II).

TABLE I

Details of 137 infants intubated for endotracheal toilet and/or oxygenation after delivery.*

From 754 operative obstetric deliveries all conducted under a standard anaesthetic technique.† (From a total of 6,574 deliveries in a 32 month period).

	Total	Infants intubated			
		Number of intubations	Pre-operative foetal distress	Failures (stillbirth)	Neonatal deaths
Caesarean section	333	80 (24%)	43 (54%)	4	6
Midcavity forceps	245	41 (17%)	20 (49%)	—	3
Outlet forceps	153	12 (8%)	8 (75%)	—	2
Breech delivery	23	4 (17%)	3 (75%)	1	1
Total	754	137 (18%)	74 (54%)	5 (3.6%)	12 (8.7%)

* According to the criteria outlined in text. † Thiopentone-suxamethonium-nitrous oxide-oxygen.

TABLE II
The reason for intubation in 137 infants.
From 754 operative deliveries.*

Primary respiratory depression at birth	54%
Secondary respiratory depression at birth	17%
Obstruction due to intrapartum, or postpartum inhalation	20%
Mixed causes	8%

* See table I.

Only in relatively few instances did the duration of intubation exceed 20 minutes, ranging in most cases from 5 to 15 minutes (Table III). Frequently only brief intermittent positive pressure inflation with oxygen was necessary before regular spontaneous respirations were initiated even in puny and depressed infants.

TABLE III
*The duration of intubation in 137 infants intubated for endotracheal toilet and/or oxygenation.**
From 754 operative deliveries.†

Percentage of total	27	38	20	11	4
Time in minutes	<5	<10	<15	<20	>20

* According to the criteria outlined in text.
† See table I.

Table IV shows the progress of the infants after delivery. Of the group of 120 live infants, 102 remained well and were healthy on discharge from hospital. Twelve infants showed transient effects of birth trauma but were also well on discharge. Six infants showed persistent residual atelectasis which cleared within 2 to 3 days. These infants were treated in an incubator and were thriving on discharge. Four of this last group had been premature by weight and five were delivered by Caesarean section. All premature infants when

TABLE IV
*Showing the subsequent progress of 137 infants intubated at birth for the purpose of endotracheal toilet and/or oxygenation.**
From 754 operative deliveries.†

	Number of infants
Failed resuscitations (stillbirths)	5
Neonatal deaths (see table V)	12
Gave no further anxiety, and discharged thriving	102
Some persistent atelectasis for 2–3 days—discharged thriving	6‡
Transient cerebral signs—discharged thriving	12
	137

* According to the criteria outlined in text.
† See table I.
‡ Five infants delivered by Caesarean section and four premature by weight.

subsequently seen in the paediatric out-patients department were progressing well. In none of the group of 120 infants was there any sequelae observed which might have been associated with endotracheal instrumentation.

Table V details the 12 neonatal deaths which took place among the group of 137 infants intubated at birth. Table VI gives details of the 10 neonatal deaths among the remainder of the 754 operative obstetric deliveries. In table V we may exclude from our considerations all but the single instance of hyaline membrane disease (pulmonary syndrome) and the 4 instances of intrapartum asphyxia. These latter infants died at 4, 6, 11 and 12 hours (the last being the resuscitated stillbirth), and death was considered to be due to atelectasis associated with intrapartum distress and aspiration of amniotic fluid and debris. Clinically treatment had improved but failed to reverse the condition. There was no evidence at autopsy (at

TABLE V
*Neonatal deaths occuring in 137 infants intubated at birth for the purpose of endotracheal toilet and/or oxygenation.**
From 754 operative deliveries.†

	Congenital abnormality	Birth trauma	Pulmonary syndrome	Prematurity	Intra-partum asphyxia	Incidental disease	Total
Caesarean section	1	—	1	1	2	1	6
Forceps delivery	2	1	—	—	2	—	5
Breech delivery	1	—	—	—	—	—	1
Total	4	1	1	1	4	1	12

* According to the criteria outlined in text. † See table I.

TABLE VI

Neonatal deaths occurring in 617 infants in whom resuscitation was not considered necessary at birth. From 754 deliveries.*

	Congenital abnormality	Birth trauma	Pulmonary syndrome	Prematurity	Intra-partum asphyxia	Incidental disease	Total
Caesarean section...	—	—	1	1	1	1	4
Forceps delivery ...	2	3	—	—	—	—	5
Breech delivery ...	—	1	—	—	—	—	1
Total	2	4	1	1	1	1	10

* See table I.

which the pharynx, trachea, and lungs were carefully examined) to suggest that tracheal instrumentation had led to any untoward sequelae.

DISCUSSION

In 1752 William Smellie wrote, "the child has been sometimes recovered by blowing into the mouth with a silver canula, *so as to expand the lungs*".* The tracheal catheter was recommended by Scheel in 1780 (quoted by De Lee) and De Lee (1924) stated that he intubated routinely all cases of asphyxia pallida, using his finger as a guide. He then performed artificial respiration by squeezing the chest or by blowing with his mouth. Flagg (1928) described a speculum for direct intubation and oxygenation in neonatal asphyxia.

Blaikley and Gibberd (1935) pioneered endotracheal intubation of the newborn in Great Britain. They insufflated the trachea with oxygen using a fine gum elastic catheter introduced under direct vision, which allowed a leak-back of oxygen outside the catheter. The pressure was controlled by the use of a reservoir bag and a manometer. The apparatus is not intended for, nor should it be used for, expanding the lungs. Misuse of this apparatus has probably been a factor in strengthening the still existing opposition to intubation of infants in general. The gum elastic catheter is potentially more traumatic than a soft endotracheal tube and, should the leak-back be obstructed, there is a greater danger of damage to lung tissue from excessive or prolonged positive pressure. However, until recently it has still been said that any method of attempting neonatal resuscitation by blowing gases into the lungs should be condemned (*Lancet*, 1951). The dangers and difficulties of endotracheal inflation have been allegedly so great that much alternative and complicated apparatus (such as cabinet respirators and phrenic nerve stimulators, etc.) have been designed for the performance of artificial respiration. We agree with Abramson (1956) that much of this apparatus is too complicated for general use.

Of late, large series of endotracheal intubations in the course of neonatal and paediatric anaesthesia have been reported, showing a very low incidence of complications (Eckenhoff, 1951; Smith, 1953; Pender, 1954; Rees, 1958). These have modified the view held previously that "special hazards are associated with instrumentation of the infant larynx". Rees (1958) regards these hazards as further examples of medical mythology. Furthermore, it is now known that positive pressure should not be held for longer than 0.5 sec and that the lungs can withstand a high pressure for a fractional period better than a more prolonged lower pressure (Day et al., 1952). Ideally, skilful endotracheal intubation is less likely to cause trauma or infection than the traditional blind pharyngeal proddings of the sucker to which many infants born in this country are subjected. Since an anaesthetist is often the specialist best acquainted with the respiratory airway the dynamics of respiration and of circulation and the necessary technical procedures, it seems good sense that he should, on occasions, assume the responsibility for neonatal resuscitative procedures (Abramson, 1956); indeed such procedures are being placed more and more under the care of the anaesthetic staff (Secher, 1956).

Naturally no direct comparison between the neonatal death rates in the intubated (table V) and nonintubated infants (table VI) can be made

*The italics are ours.

as the former group was especially selected on the grounds of respiratory difficulties. A study of these deaths is, however, not uninformative.

In our series of nonintubated infants (from 754 deliveries) there was only one neonatal death due to intrapartum asphyxia associated with the aspiration of amniotic debris (table VI). This infant relapsed after regular spontaneous respirations had been initiated at birth. Laryngoscopy produced considerable reaction. We agree with Roberts (1949) that most infants who actively object to the insertion of a laryngoscope blade probably do not need intubating, and so in this case oxygen was administered first by face mask only and later in an incubator. The infant rapidly deteriorated until death 14 hours later. Postmortem disclosed atelectasis with bronchi filled with amnoitic debris. Retrospectively we feel that more active intervention was indicated and might well have proved lifesaving.

One case of hyaline membrane disease in the intubated series was fewer than we had anticipated, for it is widely held that this condition is prone to occur in premature infants delivered by Caesarean section (Claireaux, 1954). Only one other case occurred in the whole series, of whom 333 were Caesarean deliveries, 33 per cent of these being premature by dates. Brown (1959) suggests that failure to establish regular respirations early is a major aetiological factor in this condition and Barrie and Bonham Carter (1959) suggest that the enormous pressure swings, in respiratory distress with negative phases of 20–30 cm water, are even more important. It is an interesting conjecture that rapid establishment of normal respiratory effort, initiated by early endotracheal inflation with oxygen, may be associated, in part, with the low incidence of hyaline membrane disease which we have encountered.

Adequate respiratory effort is often initiated in depressed and feeble infants by literally a few seconds of positive pressure inflation with oxygen. This only suggests, we feel, that intubation does not need to be maintained. We realize that the more conservative minded may deduce that interference was unnecessary and we do not question that many of the infants treated would have been "alright" if left alone, with maybe the traditional oxygen funnel as the sole treatment. Feeble infants have survived since Cain and Abel without active intervention and they will continue to do so, but, it is impossible to say whether some degree of permanent cerebral damage may not take place during periods of hypoxia. As Donald (1958) says, "(one) imagines their mentality being slowly reduced from that of the upper fifths to that of the lower fourths, as the seconds tick away". It is doubtful if it can ever be proved that transient anoxia can make the difference between a first-class honours degree and a simple pass. But Penfield (1954) states that considerably more than half of the patients on whom he operates for focal epilepsy would never have had a seizure but for brief anoxia or trauma at birth.

Politi (1957) wonders how many doctors would treat the apnoeic adult with the traditional stimulatory measures sometimes applied to the depressed neonate. We further wonder who would treat apnoeic adults by inactive conservative observation augmented perhaps with an oxygen funnel and what member of a modern surgical team would treat incipient respiratory arrest and cardiac failure with drugs, without assisting the respirations? We recognize that the physiological factors associated with the change-over from foetal to neonatal circulation and the reversion to primitive respiratory function brought about by central depression are absent in adult respiratory or cardiovascular failure. This does not alter the fact, that central anoxia is the primary aetiological factor and that this can be, and must be, corrected.

CONCLUSIONS

Our conclusions are merely additive to many of those already outlined (Russ, 1946; Roberts, 1949; O'Brien and Roberts, 1952; Lord, Powell and Roberts, 1953; Kromm, 1955; Flowers, 1956; Abramson, 1956; Secher, 1956; Schmidt, McLandress and Cruickshank, 1956; Shroeder, 1957; Burmeister, 1957; Donald, 1957; Politi, 1957; Rees, 1958). We hope that the publication of a further successful series, in which endotracheal resuscitative measures have been applied in the newborn may speed the general adoption of a technique which we believe has been already too long delayed.

A controlled clinical trial designed to demonstrate the worth of intubation would be extremely difficult, especially for those who believe in its value. Russ (1946), Fletcher and Rogers (1951),

Lord, Powell and Roberts (1953) and O'Brien and Roberts (1952) report series of intubations in the newborn. From their figures, and our own, it is not possible to analyze the role of intubation by the statistical method. We are, however, confident that no harm results, and like many others we regard endotracheal intubation as the safest and most effective means of resuscitation, initiating regular respiratory effort, correcting hypoxia, and of indirectly accelerating the change-over over from foetal to neonatal circulation.

Endotracheal intubation is not the only method of neonatal resuscitation, but it is the most effective. Early treatment is essential and the sooner adequate oxygenation is achieved the shorter is the duration of assistance necessary.

We consider that intubation of the apnoeic neonate is best performed with endotracheal catheter of a size similar to a 00 Magill tube. The lungs should be expanded and inflated intermittently using a pressure not greater than 40 cm H_2O and usually much less, using a very brief inspiratory phase. Some modification of the T-piece principle (Ayre, 1937) should be used to prevent rebreathing and to allow free expiration. When the lungs are expanded lower pressures suffice for artificial respiration.

We have found it relatively easy to maintain oxygenation in the majority of instances. Our greatest difficulty has occurred when there has been intrapartum aspiration, which has not responded to repeated suction so that some degree of respiratory difficulty or cyanosis persists. However, in most instances oxygenation was improved, and the general condition could be maintained by such therapy. Sometimes, however, residual atelectasis persisted for a few days.

The objections raised to endotracheal intubabation can no longer be justified. the essential requirements include experienced personnel, a minimum of equipment, and of great importance, mutual agreement and liaison between the departments concerned. It is our experience that the procedure is simple to perform when the proper facilities are at hand and when the responsibility is well defined in a department accustomed to such procedures. When the personnel is inexperienced in the technical procedure involved, and when the departments concerned are unaccustomed to the method, suspicious of its dangers, and with no clearly defined delegation of responsibility, we agree that the indications for intubation might be modified especially in the presence of other resuscitative measures. When intubation is the policy, however, the individuals responsible must be capable of rapid nontraumatic instrumentation.

Brown (1959) underlines the fact that respiratory and circulatory functions cannot be separated, but whereas improvements in oxygenation will lead to improvements in circulation, the treatment of circulatory collapse is of little avail in the presence of apnoea and the absence of oxygenation.

There are, of course, dangers to endotracheal intubation, as there are dangers associated with surgery, anaesthesia and the administration of drugs. The dangers do not, however, prevent the correct application of the technique.

SUMMARY

The factors associated with the resuscitation of a group of 137 newborn infants by means of endotracheal intubation, aspiration, and inflation with oxygen are analyzed. There was no evidence of any morbidity associated with this regime, which provides a simple, valuable, and efficient means of achieving early oxygenation, the importance of which is discussed.

We stress that the large-scale success of this procedure depends on the use of the correct equipment, favourable departmental organization and the right attitude of mind.

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SOME FACTORS ASSOCIATED WITH NEONATAL DEPRESSION IN OPERATIVE OBSTETRICS

BY

R. J. HAMER HODGES, E. J. WILSON, R. F. KNIGHT,* AND M. E. TUNSTALL

The Obstetric Unit, St. Mary's Hospital, Portsmouth, England

MANY factors have been held responsible for respiratory depression in the newborn following operative delivery. Intrapartum asphyxia, birth trauma and central depression from drugs are among the most commonly incriminated. Adequate antenatal care, careful observation of the foetal heart during labour, skilful obstetrics and the use of nondepressant analgesic and anaesthetic techniques go far to obviate many of the preventable causes. The remaining unavoidable aetiological factors are not easily segregated but recently we have had the opportunity to examine some of these factors and to draw some conclusions therefrom.

MATERIAL AND METHOD

During a 32-month period 6,574 deliveries took place in the obstetric unit concerned (table I). Of these 873 (13.3 per cent) were operative deliveries under anaesthesia. Of the operative deliveries 119 patients (13.6 per cent) received general anaesthesia likely to affect neonatal respiratory activity (Apgar et al., 1957; Sjostedt and Rooth, 1958; Roberts, et al., 1957; Kolstad and Schye, 1957; Donald, Kerr and Macdonald, 1958; Hodges et al., 1959) and these were excluded. The remaining 754 deliveries were conducted under a standardized general anaesthetic technique, which has been shown to have minimal effects on the newborn (Hodges et al., 1959). Of the infants in this latter group, 137 were intubated at birth for the purposes of aspiration or oxygenation (Hodges et al., 1960).

Records were kept of the pre-operative obstetric conditions prevailing, the state of the infant at birth and the subsequent progress. The special anaesthetic-obstetric record card already described (Hodges, 1959) was used. The association of anaesthetic and obstetric factors with the incidence of postpartum depression was examined and analyzed. In particular it was desired to determine the incidence of pre-operative foetal distress and the effects of timing of pre-operative analgesic drugs.

The circumstances in which postpartum respiratory difficulties are most likely to arise are outlined.

RESULTS

Firstly, it was necessary to exclude as far as possible any variable depressant effect of the anaesthetic technique used.

*Present appointment: Captain, R.A.M.C.

TABLE I

Total number of deliveries	Number of operative deliveries (873)		Number of infants intubated in the standard anaesthetic series	Operative procedure
	Nonstandard	Standard		
6574	29	333	80	Caesarean section
	40	245	41	Midcavity forceps
	40	153	12	Outlet forceps
	10	23	4	Breech delivery
Totals	119	754	137	

It has already been shown that, of operative deliveries, those infants whose mothers received thiopentone-suxamethonium and nitrous oxide-oxygen appeared to breathe and cry more readily at birth and to have less postpartum depression than those whose mothers were anaesthetized by more traditional techniques (Hodges et al., 1959).

In 754 patients (Caesarean sections and forceps—see table I) the results of the use of this technique, standardized in this unit, was examined to see whether, apart from being apparently the best of the compared methods, it was in fact nondepressant to the infant over a larger series of patients. It will be seen from figure 1 that, when all patients were considered, two-thirds of the infants delivered were breathing regularly within 30 seconds of delivery and over 90 per cent of all infants did so within 5 minutes.

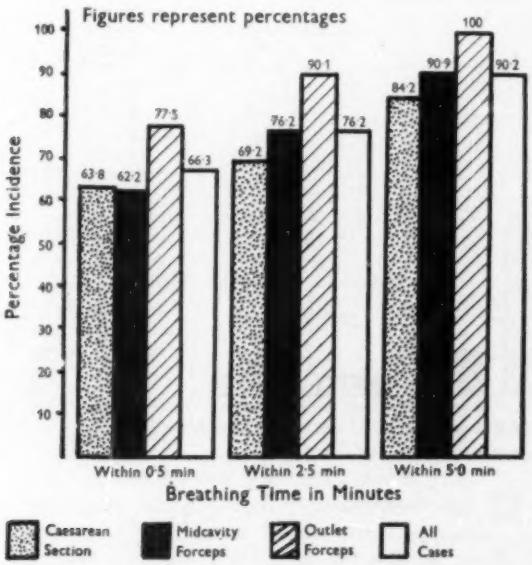

FIG. 1

The incidence of infants who breathed regularly within 0.5, 2.5, and 5.0 minutes—from 754 patients* delivered under general anaesthesia.†

*Caesarean section 333. Midcavity forceps 245. Outlet forceps 153. Breech deliveries 23. (Stillbirths excluded.)

†Thiopentone-suxamethonium-nitrous oxide-oxygen.

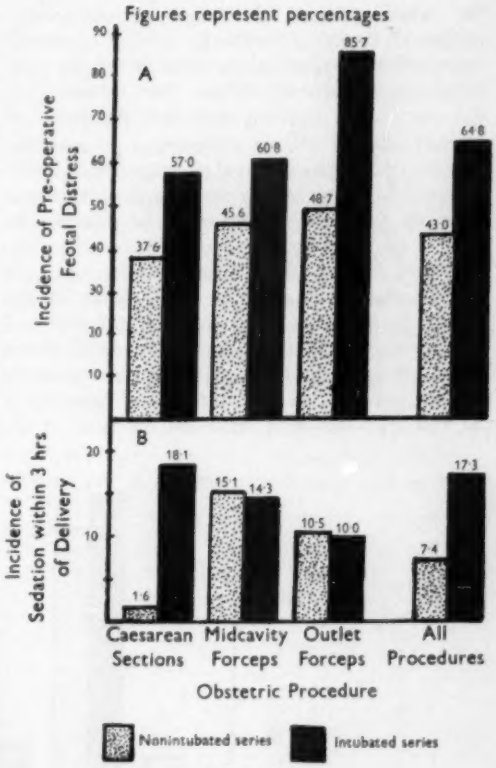

FIG. 2

The incidence of (*a*) pre-operative foetal distress and (*b*) the administration of maternal sedative drugs before delivery, in 731 infants of whom 133 were intubated after delivery for the purposes of endotracheal toilet and/or oxygenation* (all patients delivered under general anaesthesia†).

*According to criteria outlined in text.

†Thiopentone-suxamethonium-nitrous oxide-oxygen.

Fig. 2a shows the incidence of pre-operative foetal distress in 731 patients considered. The group of 23 breech deliveries (table I) was excluded from the series as the number was too small for adequate analysis. It will be seen that the incidence of foetal distress pre-operatively is uniformly higher in all types of delivery where respiratory difficulties necessitated endotracheal intubation. In Figure 2b the administration of pre-operative drugs to the mother is considered in the same series. It will be seen that, taking all infants, there was an increased incidence of

the administration of drugs pre-operatively (within 3 hours of delivery) in the intubated series, 17.3 per cent compared with 7.4 per cent in the nonintubated. When this is analyzed, however, it is apparent that this difference is entirely due to infants delivered by Caesarean section, where the incidence of drugs administered within 3 hours to infants needing intubation was over ten times the incidence in the infants who needed no resuscitation.

Figure 3 shows the interval between the start of induction of anaesthesia and delivery of the infant in its relationship to the intubated and nonintubated infants. This figure overall shows little difference in the two series, substantiating the suggestion that the duration of anaesthesia of this type has little effect on the state of the infants at birth. Closer examination of the figure, however, discloses some small differences from which interesting implications may be drawn. It will be seen that an induction-delivery interval of more than 25 minutes occurred 3½ times more often in the intubated series than in the nonintubated, whereas in the 20–25 minute interval no difference between the two series is apparent. This, it is believed, is a reflection of the respiratory depression encountered when obstetric difficulties produce a marked delay in the delivery of the child. The aetiological factor in respiratory depression in this group is probably unavoidable birth trauma associated with obstetric difficulties.

Further, from this figure, it is seen that of the infants requiring intubation, a lower percentage

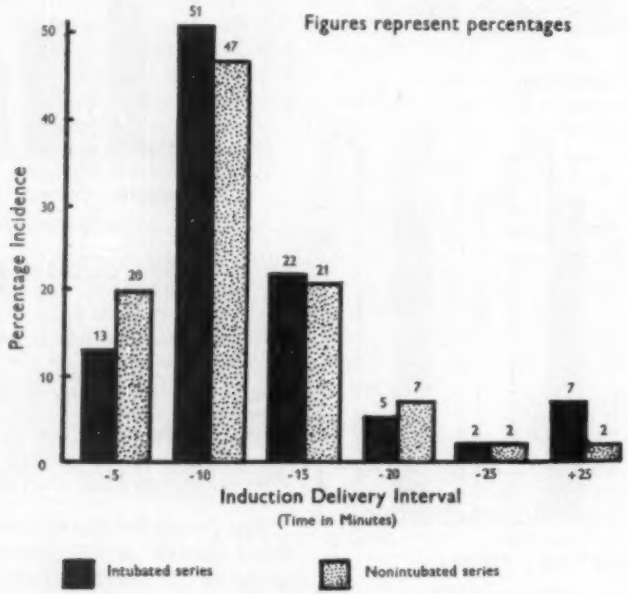

FIG. 3

The interval between induction and delivery in 754 operative deliveries,* 137 infants being intubated after delivery for the purposes of endotracheal toilet and/or oxygenation† (all patients delivered under general anaesthesia‡).

* Caesarean section 333. Midcavity forceps 245. Outlet forceps 153. Breech delivery 23.

† According to criteria outlined in text.

‡ Thiopentone-suxamethonium-nitrous oxide-oxygen.

(compared with the series who did not require intubation) fell into the 0–5 minute induction-delivery interval, and a higher percentage into the 5–10 minute interval.

To investigate this further, the relationship of the breathing time to the induction-delivery interval was examined in 333 Caesarean section infants (table II). It will be seen that the incidence of infants with a breathing time of 5 or more minutes was maximal in the 7–11 minute induction-delivery interval, with the exception of those infants whose delivery had been exceptionally delayed (more than 15 minutes). The implications of these findings are discussed later.

thesia. As the technique was standardized throughout the series it is also possible to draw conclusions from the incidences of the other factors considered.

Influence of anaesthesia.

The writers' opinion (Hodges et al., 1959) that an anaesthetic technique based on thiopentone-suxamethonium-nitrous oxide-oxygen affects the infant to a minimal degree is further substantiated, and it is considered that any such effect can be disregarded in practice. However, the results again suggest that (when those deliveries in which technical difficulties have been experienced are excluded) it is the infants delivered in

TABLE II

The relationship of the breathing time to the induction-delivery interval in 333 *Caesarean born infants.**
Showing the percentage incidence of infants with breathing time of 5 minutes or more in each of the induction-delivery periods.

Breathing time (minutes)	Induction delivery interval—in minutes (Number of infants in each group)									Totals
	<1	<3	<5	<7	<9	<11	<13	<15	>15	
≤1	–	10	70	59	32	25	13	3	7	219
≤2	–	–	4	3	3	2	1	2	3	18
≤3	–	–	2	2	1	–	–	–	–	5
≤4	–	–	1	3	–	1	2	–	3	10
≤5	–	–	2	1	1	1	1	1	2	9
≤6	–	–	3	2	4	1	–	–	2	12
≤7	–	–	–	1	2	–	–	–	1	4
≤8	–	–	2	3	4	2	–	–	1	12
≤9	–	–	2	1	–	1	–	–	–	4
≤10	–	–	1	1	–	2	–	–	2	6
>10	–	1	4	3	2	6	2	–	2	20
Totals	–	11	91	79	49	41	19	6	23	319
Number in each group with breathing time 5 minutes or more	– 0%	1 9.1%	12 13.2%	11 13.9%	12 24.5%	12 29.3%	2 10.5%	– 0%	8 34.8%	58 18.6%

* All conducted under thiopentone-suxamethonium-nitrous oxide-oxygen excluding stillbirths.

DISCUSSION AND CONCLUSIONS

Barrie and Bonham Carter (1959) state that 10 per cent of all liveborn infants that they encounter have regular respirations delayed for more than 5 minutes after delivery. In the present series of 754 operative deliveries no more than 10 per cent were so delayed (fig. 1). It is considered therefore, that the anaesthetic technique in use has a minimal and negligible effect on infant respiratory activity. However, it is felt reasonable to draw certain conclusions regarding the effect on the infant of this method of anaesthesia.

the 7–11 minute induction-delivery interval who are most affected. It is repeated, therefore, that, contrary to the opinion of others (Crawford and Kane, 1956; Steel, 1957), a rapid delivery of the infant may still have a definite advantage, especially in the presence of foetal distress. Speed should not, of course, take precedence over safety.

From our results there appear to be three factors which significantly predispose to the occurrence of postpartum neonatal depression when the effects of toxic depressant anaesthetic agents (ether, cyclopropane, trichloroethylene)

have been excluded. These factors are: the administration of sedative drugs pre-operatively to the mother; the presence or absence of foetal distress in the first instance; and a prolonged induction-delivery interval associated with obstetric difficulties.

The influence of pre-operative foetal distress.

In all women in whom Caesarean section or forceps delivery is undertaken for foetal distress, the incidence of postpartum depression of the infant rises, irrespective of the type of operative procedure. This is a consideration which should seriously concern the anaesthetist, for not only are more than 50 per cent of the total anaesthetic calls for operative obstetrics associated with foetal distress in our experience, but further the responsibility for resuscitative procedures in the newborn is falling increasingly on to the anaesthetic staff (Secher, 1956; Hodges et al., 1960).

Influence of analgesic drugs administered pre-operatively.

In those patients to whom sedative drugs have been administered less than 3 hours pre-operatively there is an increased risk of neonatal depression. It is in those women delivered by Caesarean section in whom this added risk is most dramatically illustrated. It would appear advisable that in elective procedures, operations should be postponed where sedation has recently been administered and the routine administration of "pre-operative" sedation must be regarded as a hazard to the infant.

Other factors.

Factors which did not appear to have any influence on the necessity or otherwise for endotracheal intubation of the infant after delivery included prematurity of up to 4 weeks (though the incidence of depression rose in infants of less than 36 weeks gestation), pre-operative toxaemia of pregnancy, and the age of the patient. The primigravida and the grand multiparae do not, from our results, appear to be affected in this respect.

SUMMARY

Factors associated with postpartum respiratory difficulties in the newborn are examined in a series of 754 operative obstetric deliveries in which 137 infants were intubated after delivery for the purposes of aspiration or intermittent positive pressure inflation with oxygen.

The previous conclusion (Hodges et al., 1959) that an anaesthetic technique based on thiopentone-suxamethonium and nitrous oxide-oxygen is nondepressant to the infant was further substantiated.

The incidence of pre-operative foetal distress causes an overall increase in infant respiratory difficulties irrespective of the operative procedure, be it Caesarean section or forceps delivery. The administration of drugs less than 3 hours before delivery greatly increased the hazard of postpartum respiratory depression in those infants delivered by Caesarean section. The infants in whom for obstetric reasons delivery was delayed for more than 25 minutes after anaesthesia was induced also showed an increased incidence of respiratory difficulties, probably associated with birth trauma.

Other factors appeared to be of little significance.

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AN UNUSUAL CAUSE OF AIRWAY OBSTRUCTION

A Case Report

BY

C. P. BAHL

Irwin Hospital, New Delhi, India

THERE are many causes for obstruction of the airway above the glottis. The case discussed below is interesting in that this complication originated from the presence of a roundworm in the pharynx. Roundworms have been known to be expelled in the vomitus, but to the author's knowledge there is no case discussed in the anaesthetic literature where the presence of a worm above the glottis resulted in respiratory obstruction.

CASE REPORT

I. J., 26 years, male, was brought to the hospital on August 17, 1959, with acute appendicitis of 3 days duration. He was scheduled for emergency appendicectomy on the same day. Subarachnoid block was undertaken using 1.6 ml cinchocaine (1:200). This was followed by thiopentone 300 mg in 2.5 per cent solution, nitrous oxide, oxygen and minimal ether. Trichloroethylene was used to assist the uptake of ether. Suction through a Ryle's tube before general anaesthesia yielded only a small quantity of fluid.

The operation was commenced but it was observed that the patient was becoming cyanosed. The nitrous oxide was therefore cut off and oxygen alone was administered but cyanosis increased and signs of obstruction of the airway were apparent. Laryngoscopy revealed a roundworm curled up in the pharynx which was removed by a Magill's forceps. There was no mucus, or vomited material in the pharynx. Intubation was then performed and light general anaesthesia was continued with nitrous oxide, oxygen and ether. The subsequent course of anaesthesia was uneventful.

At laparotomy roundworms were palpated in the small gut. The appendix was inflamed. At one place, the wall of the gut was damaged, so omentum was stitched over it, and the abdomen closed.

DISCUSSION

In the case described the cause of respiratory obstruction was a roundworm above the glottis. How it reached there is not clear. It could have been brought there by the movements of the worm itself, or by retching, vomiting or straining. The induction of anaesthesia was smooth and vomiting did not occur. Movements of the worm itself, assisted presumably by the contractions of the gut due to subarachnoid block or some straining which went unnoticed, may be the factors concerned in bringing the roundworm above the glottis in this case.

SUMMARY

A rare, probably unique, case of obstruction to the airway by a roundworm above the glottis is described.

ACKNOWLEDGMENTS

My thanks are due to Dr. P. Diesh, Medical superintendent, Irwin Hospital, New Delhi, for permission to publish the case.

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INFANT PHYSIOLOGY AND ANAESTHESIA

BY

IAN H. MCDONALD

Department of Anaesthesia, Royal Children's Hospital, Melbourne, Australia

Oliver Wendell Holmes said:

So the stout foetus, kicking and alive,
Leaps from the fundus for his final dive,
Tired of the prison where his legs were curled,
He pants, like Rasselas, for a wider world.
(*Wendell Holmes*)

And in the next few moments occurred a revolution in his physiology which decided the nature of his early existence.

It is convenient to divide early life into three compartments:

(1) Prenatal and natal period.
(2) Neonatal period of about two months during which the physiology is making a comparatively rapid change to a reasonably stable condition.
(3) The babe and child whose physiology has reached a phase where changes take place slowly.

PRENATAL AND NATAL PERIODS

There has been a good deal of philosophy and experimentation devoted to the discovery of the mechanism leading to the first vital breath. Snyder and Rosenfeld (1937) said that this was a continuation of rhythmic intra-uterine respiratory movements, and that movement of amniotic fluid was necessary for the normal development of the pulmonary alveoli. That this is incorrect is evident from the normal histological structure of sequestered lobes. Barcroft's work is at present widely accepted and in his work with sheep embryos he has shown a progressive change in the nature of respiratory movements exhibited by the ageing foetus (Barcroft, Kennedy and Mason, 1940).

If the foetal respiratory centre has developed along the lines indicated by Barcroft it will be capable, at term, of responding with spontaneous rhythmic efferent impulses to the usual afferent stimuli of birth. This is the state of pneumotaxis. The centre will already have passed through the developmental phases of gasping and apneusis (intermittent rhythm) which characterize earlier, less specialized periods of development. On the other hand the respiratory centre of the premature infant is still at an earlier level of function and the initial efforts at respiration may be less than optimal.

Similarly, the infant damaged by placental insufficiency, maternal disease, severe birth trauma, prolonged hypoxic labour, or heavy maternal narcosis has a less effective respiratory centre whose initial efforts may be limited to apneusis or even gasping. The subsequent prognosis of the baby depends on the severity and permanence of the medullary damage; the severely damaged brain and that of the very premature foetus cannot make the step up from apneusis to pneumotaxis and the baby will gradually pass down through the phases of apneusis, gasping and eventually to death. On the other hand the temporarily anoxic or depressed medulla (provided oxygenation is assured) will at greater or less speed develop the rhythmic respiratory pattern of normality. Unable perhaps to respond to the normal mechanical stimuli of birth, such a medulla may fortunately respond to the chemical ones of hypoxia, excess carbon dioxide or both, and although the first breaths may be gasps, these will give place to apneustic rhythm and finally to normal respirations. As Barcroft says, "The dying foetus in its last effort opens the door to life".

Early effective oxygenation will establish normal medullary function most quickly, but the more obvious methods of doing this are sometimes poorly carried out. It is worth re-emphasizing the positive virtues of clearing the upper airway, not only by suction, but by inclining the

baby's head and face downwards, when up to 40 or 50 ml of liquor may on occasion be drained out. This may be an unhurried procedure as the neonate seems capable of surviving up to about 15 minutes apnoea, though such a period of delay is obviously undesirable.

That the infant is able to survive up to 15 minutes anoxia is due to two factors. Firstly the cortex, underdeveloped at birth, is less dependent upon oxygen than the more complex organ of a few weeks later. More importantly, the neonate shows a capacity for anaerobiosis, demonstrated particularly by anaerobic glycolysis in which the cycle terminates at the pyruvic acid level.

More active methods of resuscitation involve the use of oxygen by intranasal or intragastric routes, or by the provision of intermittent positive pressure ventilation. I suspect that the infrequent use of the latter method is related to the irregular attendance at deliveries of trained paediatric anaesthetists, but at the same time it is true that sometimes positive pressure is quite ineffective in expanding previously unexpanded alveoli.

Whereas it is said that the baby uses a suction pressure of only 30 mm Hg during the first few inspirations when the alveoli are distending, a pressure of 300 or 400 mm Hg, some ten times as much, applied briefly through an endotracheal tube will often fail to expand the lungs lying free in the open chest if they have been collapsed for a day or two after birth, due to hyaline membrane disease or to the pressure of a large diaphragmatic hernia. Under these conditions the anaesthetist is quite unable to produce alveolar expansion by the normal method of bag squeezing, though Day et al. (1952) have successfully used extremely high pressures repeated at very brief intervals to do so. Not only does bag squeezing fail, but in fact it increases deadspace, while the prolonged inhalation of dry gases certainly is detrimental and may serve to establish a hyaline membrane more severely. The recent introduction of the narcotic antagonists seems to have rendered a signal service to the baby, their use either prenatally for the mother or postnatally in the baby being efficacious.

At the same time as the respiratory centre is developing and the foetus is taking its first "non-parasitic" breaths, the circulation is undergoing changes which, though of less immediate importance as far as the anaesthetist is concerned, throw more light on the ability of the neonate to survive quite long periods of anoxia.

Oxygenation of the foetus is dependent upon the following factors:

The efficiency of the placenta as a gas transferring medium.
The nature of the foetal haemoglobin.
The amount of circulating haemoglobin.
The amount of foetal blood circulating through the placenta.

As the foetus approaches term the placenta becomes comparatively less able to provide complete oxygenation and the umbilical vein blood oxygen saturation falls from about 75 per cent near term to about 60 per cent at term and 30 per cent after term. But at the same time, perhaps in response to the relative tissue hypoxia, the haemoglobin concentration is rising to about 17 grams per cent at term, so that in terms of volumes per cent the infant at term has a reasonably high umbilical vein oxygen content of 12.5 volumes per cent, but at 42 to 43 weeks this has fallen to 8 volumes per cent.

Moreover foetal haemoglobin is formed until term. There is a shift of the oxygen dissociation curve to the left with foetal haemoglobin so that there is a 75 per cent saturation of haemoglobin at an oxygen tension of 40 mm Hg as compared with 50 per cent saturation of adult haemoglobin at 40 mm Hg which is the usual oxygen tension at the placenta.

The neonate too has a low pH of its tissues due to its relative anaerobic glycolysis; there is therefore easier unloading of oxygen.

Despite these advantages of high haemoglobin, foetal structure of haemoglobin and low tissue pH, obviously as a result of the factors of inefficient placental transfer of oxygen and mixture of umbilical vein blood with non-oxygenated blood, the foetus is accustomed to comparatively poor oxygenation at term. No doubt this factor bears a relationship to the ability of the neonate to survive rather long periods of apnoea or cyanosis, either at birth or subsequently during the first few days of life.

At term the erythrocyte count is 5,500,000 per cu.mm, but the cells are large and contain

more haemoglobin than mature cells. The haemoglobin concentration is about 17 grams per cent, falling rapidly as foetal haemoglobin is destroyed to 11 grams per cent, then rising slowly after a few weeks to reach 14 grams per cent at the age of 2 years. There is a leucocytosis of 45,000 per cu.mm, and a prolonged prothrombin time, greatest at 4 days, but prevented by the administration to the neonate of 1 mg vitamin K.

It seems certain that there is crossing of the inferior vena caval and superior vena caval streams in the foetus, but, as term approaches, more and more blood flows through the pulmonary circuit, so that the flow through the ductus arteriosus is gradually tapered off. The process is rapidly concluded at birth so that within 3 hours there is functional closure of the ductus arteriosus, ductus venosus and the foramen ovale with a consequent rapid rise of oxygen saturation from 50 to 90 per cent. The final closure of the ductus arteriosus may be related to high levels of blood oxygenation and it is conceivable that a patent ductus arteriosus may be due to a period of neonatal hypoxia.

Because of the inter-relationship of the systemic and pulmonary circulations, the ventricular pressures at birth are virtually equal and the right myocardium is as developed as the left, the electrocardiogram showing right ventricular preponderance. At birth the systemic blood pressure (as measured by a cuff 2.5 cm in width) is about 80/45 mm Hg and rises 10 points in 24 hours, with a further rise of the systolic pressure to about 95 mm Hg in 14 days. The pulse rate is variable between 90 and 180/min (average approximately 125/min).

Transient cardiac murmurs are present in about one-third of newborn infants, but significant murmurs may not be established till later.

To summarize, the infant at term has a cardio-respiratory-metabolic system designed to function adequately under the relatively hypoxic conditions of late intra-uterine life. Provided interference with these functions is minor and reversible, the newborn infant quite rapidly adjusts itself to a nonparasitic life. If very active or heroic methods of resuscitation are needed it is likely that brain damage or cardiac abnormality is of such severity as to make it unlikely that life will be prolonged.

NEONATAL PERIOD

During the next two months or so the physiology of the neonate is being adjusted to new conditions.

At the foetal age of 7 months the premature baby has no functional alveoli, the terminal bronchioles ending in blind tubules lined with cuboidal cells. The respiratory surface is therefore very small indeed. By term, the alveolar structure is fully developed, but the functional capacity of the lung remains at a comparatively low level as compared with the adult. In fact in comparison with the adult the newborn at term has half the respiratory exchange surface per unit lung weight, and one-third the respiratory exchange surface per unit body weight. Moreover, the metabolic rate shortly becomes comparatively high. The baby has therefore a rapid respiratory rate to compensate for these deficiencies. Respiratory rate and volume show considerable variation from case to case and time to time, but average figures determined by Cross (1949), Deming and Hanner (1936), and others indicate that the neonate breathes a tidal volume of 16 to 20 ml about 35 times a minute, and so has a minute volume of the order of 500 to 700 ml. During crying the tidal volume can increase beyond 160 ml. As compared with the adult, therefore the baby shifts about twice as much air to provide an equal gaseous transfer. Whilst it can cope with this under normal conditions, an increased demand for oxygen or for clearance of carbon dioxide involves comparatively large increases in tidal and minute volumes because of the relatively large physiological deadspace of the infant. Bearing in mind that the infant trachea and bronchi are about one-quarter the diameter of those of the adult, it is apparent that increased respiratory work can throw a severe strain on the respiratory musculature, which is already acting at a disadvantage because of the horizontal position of the infant's ribs (fig. 1). This is well demonstrated by the young baby who, though not obstructed, shows suprasternal and intercostal retraction whilst breathing deeply and rapidly, as a result of the inability of the air passages to pass air at a rate sufficient to keep up with the rapidly moving diaphragm.

Always remembering how important it is to

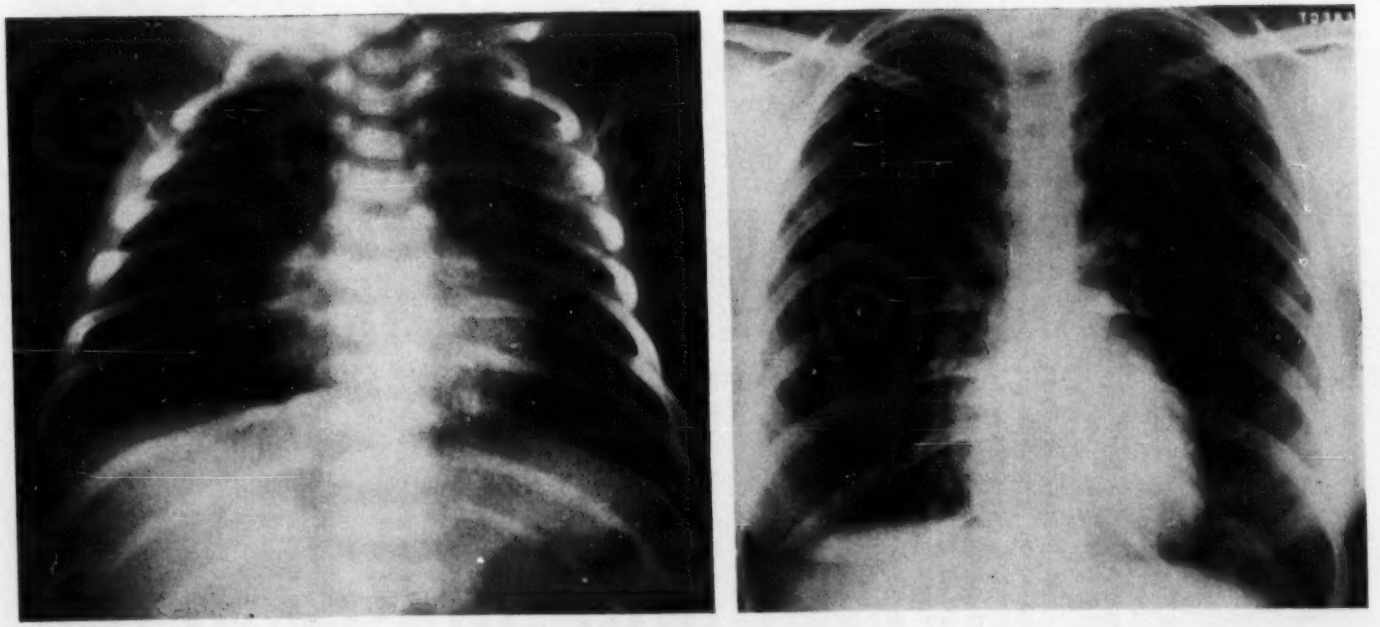

FIG. 1
Radiographs of neonate (left) and adult (right) chests, illustrating comparative anatomy of ribs.

prevent any unnecessarily increased respiratory effort the anaesthetist must avoid:

(*a*) Any carbon dioxide retention.
(*b*) A rise in body temperature.
(*c*) Any obstruction to the respiratory efforts, as, for example, by surgical assistants leaning on the chest.
(*d*) The use of apparatus with a high resistance to respiration.

Endotracheal tubes with diameters as large as will readily pass the cricoid ring, high gas flows, and T-pieces instead of valves, are necessary. The avoidance of coverings and even hot water bottles in summer is an often neglected but obviously necessary measure. An increase in weight leaning on the chest of about 10 kg doubles the respiratory work of an infant breathing spontaneously.

In regard to the respiratory anatomy of the baby the following points are of practical importance.

(*a*) The length of trachea in the newborn is about 2.5 cm, increasing in the adult to 10 cm.
(*b*) The optimum length of endotracheal tubes (i.e. distance from the lips to above the carina) can be calculated as (age ÷ 2) + 12 cm. Endobronchial intubation is all too common.
(*c*) The diameter of the trachea in terms of Magill tube sizes = (age ÷ 2) + 1.
(*d*) The cricoid ring is the narrowest part of the trachea and larynx.
(*e*) The infant larynx is situated relatively far forward in relation to the pharynx.
(*f*) The epiglottis is a slippery, thin, rolled-up structure.

With these points in mind the anaesthetist intending to intubate the neonate will provide himself with a tube of the right length and diameter (of which there are many varieties, the Oxford right-angled being a good choice for general purposes), an infant laryngoscope with a wide beak to restrain the slippery epiglottis (Flagg, Lundy, or Guedel patterns), and an assistant who will gently push the larynx backwards into the field of vision of the laryngoscope. Gentle intubation is essential bearing in mind that a tube which is too wide may render the mucosa in the region of the cricoid ring ischaemic, and that about 2.5 cm past the larynx a tube which is too long will enter the right main bronchus. Swift intubation is also required, remembering the baby's high rate of oxygen utilization and rapid onset of cyanosis. Furthermore, the respiratory epithelium is extremely sensitive to irritation and extubation can give rise to a severe prolonged bronchospasm, particularly following cyclopropane anaesthesia. This can be greatly minimized by removing the tube quickly from a well oxygenated baby at the end of a deep inspiration.

During these first few weeks the main circulatory change is the drop in normal haemoglobin value from 17 grams per cent to 11 grams per cent as the foetal haemoglobin is destroyed. The blood volume at birth is nearly 120 ml/kg of body weight, but this soon begins to fall and a normal value in a few months is about 90 ml/kg.

The maintenance of a normal blood volume and electrolyte level, and the control of drug concentrations is, in these early weeks, precarious because renal function is comparatively poorly developed, particularly in the first week. Both glomerular function and tubular resorption are inefficient and the kidney does not deal effectively with an excess water load. Because of the inability of the baby to deal with excess water and salt, intravenous therapy is dangerous unless well controlled. Together with a low phosphate excretion in the first few days there is a low serum calcium level and this combination, particularly if associated with a degree of respiratory alkalosis from overbreathing, can bear some relationship to tetany occasionally seen in the anaesthetized baby.

Two neurological factors of considerable importance are the general irritability of the cortex in the young, and the lack of temperature control in the baby, particularly the premature baby. Cortical hyperexcitability is manifested by convulsive seizures, often induced by the toxaemia of a pyrexial infection. Most convulsions under anaesthesia occur in such circumstances, particularly if associated with some hypoxaemia. The immediate treatment consists of full oxygenation, if necessary combined with muscular paralysis and intermittent positive pressure breathing.

The lack of an effective temperature-controlling mechanism in the young baby combined with its relatively large surface area, can produce rapid and severe temperature changes. This is particularly dangerous in hot weather where the baby is delivered to the theatre in the time-honoured fashion, swathed in blankets. Common-sense nursing and the limitation or omission of pre-anaesthetic atropine can effectively prevent what is not uncommonly a fatal hyperpyrexia. Conversely in cool weather, falls in rated temperature of the order of 95°F (35°C) are not uncommon, and during long operations should be countered by the use of a warm water mattress.

Into the neonatal age group fall particularly the anaesthetics involved in the surgery of congenital obstructions of the gut from the mouth to the anus, diaphragmatic hernia, exomphalos, expanding pulmonary cysts, and some meningomyelocoeles. Anaesthesia will be almost invariably with nitrous oxide or cyclopropane given by endotracheal catheter, utilizing if necessary suxamethonium either intravenously or intramuscularly to supplement the gaseous anaesthetic. A standard tube and bag arrangement is used with a high gas flow and without an absorption canister, the open-tailed bag forming the distal end of the expiratory limb of a T (Rees, 1950). Flow rates must be related to the neonatal minute volume of about 500–700 ml and will therefore be of the order of 1½–2 l./min. (table I).

TABLE I
Gas flow rates in T-piece circuits

	Tidal volume (ml)	Respiratory rate	Minute volume (ml)	Gas flow rate (litres/min)
Neonate	16	35	560	1¼
12 months	50	30	1500	4
3 years	125	20	2500	7
8 years	250	16	4000	10
Adult	500	14	7000	20

Paralyzing drugs have the virtue of lowering basal metabolic rate considerably, while at the same time the work of respiration must be taken over by the anaesthetist, but they must be used realizing that the competitive inhibitors are poorly tolerated by the infant, whereas the suxamethonium group are adequately destroyed. The dose of suxamethonium should be limited in the neonate to a total dose not exceeding 40 mg because of the liability to prolongation of apnoea. It is of course unwise to use suxamethonium by the intramuscular route should the infant's circulation be less than good, while destruction of the drug will be prolonged should the baby's temperature fall. One of the most common and potent causes of poor postoperative breathing by the neonate is cold, whether muscle relaxants have been used or not, and every effort should be made to keep the baby's temperature normal.

Deviations from normal pH due to respiratory effects are not more easily produced in the baby than in the adult. Alkalosis may be attended by tetany (particularly in the neonate with low serum calcium and high phosphate) and by cerebral vasoconstriction (with its possible sequel—convulsions). Acidosis will put a load on the kidneys which are unable to deal with excess acid production because of their low efficiency in dealing with the phosphate ion. There must be a constant striving, therefore, to respire the patient at normal rates and volumes.

Intravenous infusions of water and blood must be controlled with extreme care to avoid overloading the circulation. At the end of the first week of life the 7 lb (3.5 kg) infant's blood volume is about 12–14 oz (350–400 ml). His oral fluid intake is nearly 1 oz (28 ml) an hour, but intravenous fluid is required at only 60 per cent of this rate. In general 5 per cent dextrose with distilled water or one-eighth normal saline is the best infusion.

THE SMALL CHILD

After 2 or 3 months physiological functions are approaching a steady level, and renal excretion allows more liberal use of curare-like drugs. In the young, rectal narcosis may be useful, but soon intravenous medication becomes more and more a standard procedure. T-pieces give way after a few years to valves and the techniques used may approach closely to those for the adult. Much of the work in this group is concerned with reconstructions and excisions about the head and neck, and herein lies a field where spontaneous respiration with nitrous oxide and ether through a T-piece is a very adequate technique.

Controlled respiration, circle-absorption, and all the adult methods are applicable in young children in abdominal and thoracic anaesthesia, provided always that the anaesthetist remains acutely aware of the finer margins for error allowable in these small patients.

SUMMARY

Infant physiology as it pertains to anaesthesia is summarized. Certain basic physiological data of practical importance in the efficient management of anaesthetized children are presented.

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A DISPOSABLE CANNULA

BY

T. M. YOUNG

Chase Farm Hospital, Enfield, Middlesex, England

APPARATUS for transfusion needs to be simple to set up, and reliable, continuing to act despite movement of the part. Drips through indwelling polythene tubing have an attractive longevity but their truly aseptic placement is time consuming. These requirements seem best answered by the needle-cannulae—West Middlesex, Frankis Evans', and Guest's. Unfortunately, one cannot always ensure the return of the cannula moiety, and we have been left with a number of half sets. The recent introduction of a disposable transfusion set avoids this problem but, to date, only a simple needle which easily cuts out of the vein has been supplied with it.

Each of a small stock, related to daily demand, of the disposable needles is paired to a long (4½-inch) thin (20 BWG) aspirating needle which just fits inside it. The giving set needle is ground

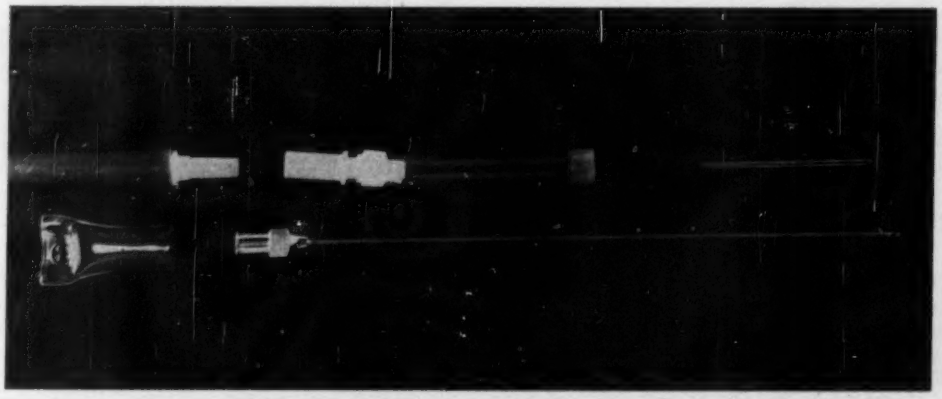

FIG. 1

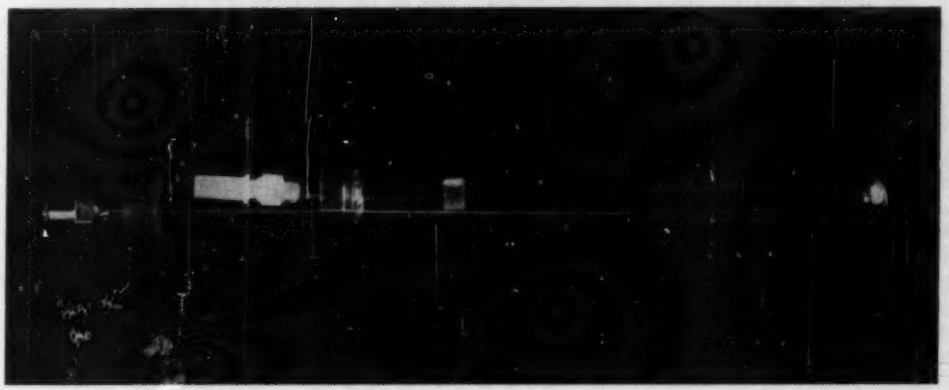

FIG. 2

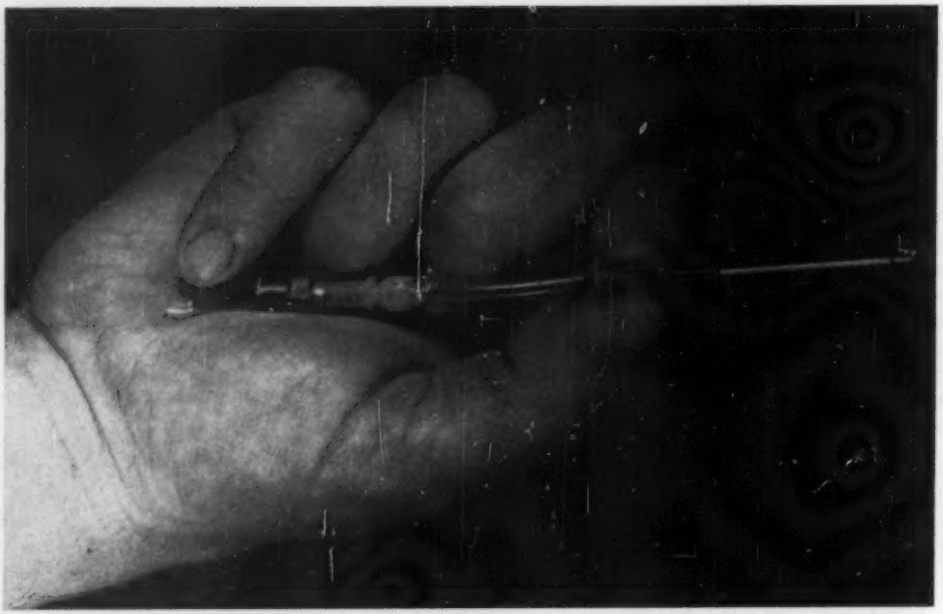

FIG. 3

square at the end to form a cannula of such a length that the needle placed within it just protrudes. The cannula is then chamfered to a round shoulder for ease of insertion (fig. 1). Now unsterile, it is replaced in its glass tube, the rubber cap put on, and the paired needle stuck through this for storage (fig. 2). Before use the needle and cannula are reassembled and sterilized by boiling. Such a long thin needle is rather whippy. To pierce the tissues there, it is essential to grasp the base of the cannula keeping the needle point protruding by pressure of the palm and little finger. The cover of the air inlet of the transfusion set when fitted over the hub of the needle maintains sterility and minimizes escape of blood during insertion (fig. 3). Clot, once formed in the needle, is well nigh immovable. It is therefore washed out as soon as possible. We use a rinse solution made up as follows: green soft soap, 25 per cent; methylated spirit, 25 per cent; sodium hexametaphosphate, 4 per cent (or q.s.); glycerin, 12 per cent; water to 100 per cent (Grainger, 1956).

The clean needle is then returned with the unused giving set needle to the instrument technician who fashions another needle-cannula pair to replenish the stock. The possibility of presenting the whole ready sterile from the Central Syringe Service is being examined. The cost of the assembly is: aspirating needle, 9*d.*; giving set needle, 1*s.* allowed on return. Technician's time, less than 5 minutes.

Recent catalogue prices of needles are appended:

West Middlesex cannula	8*s.* 0*d.*
Guest's cannula	6*s.* 6*d.*
Frankis Evans' cannula	5*s.* 0*d.*

After five transfusions these cannulae are therefore cheaper—unless they are lost. Freedom from worry over their return, and constant availability, are the advantages of the system.

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THE EVOLUTION OF ANAESTHESIA

1. ETHER IN MEDICINE BEFORE ANAESTHESIA

BY

ENID M. SLATTER

Nuffield Department of Anaesthetics, University of Oxford, England

THE anaesthetic properties of ether now quite obscure its other medicinal qualities, but, during the hundred years before 1846, it had been extensively used for a variety of therapeutic purposes. Ether became established as a medicine in the mid-eighteenth century, being principally recognized as a sedative and antispasmodic. As it is a liquid with a high rate of evaporation, when dabbed on the skin it produces a sensation of great coldness and this property was turned to good account, being especially applicable for alleviating every species of ache, "headachs and meagrims" being "dissipated instantaneously and as it were by enchantment". Ether vaporizes at room temperature and so can easily be inhaled, an alternative way of taking it for these complaints and also a means of relieving palsies, obstinate hiccups, asthma, whooping cough, convulsions, etc., etc. Taken in the form of a draught in water, lemon-juice or wine, it was good for "Flatulant Cholics" and "all windy Disorders of the Stomach". It was, in fact, a regular panacea and, since it was "perfectly innocent and safe to take, as it contains nothing that is acrimonious or corrosive", ether could also be given "even to the youngest Children without Hesitation". If this is not sufficient recommendation of its usefulness, it must be added that ether also "takes spots of grease from silks without affecting their colors".

Some of these uses are advocated by Matthew Turner, chemist and surgeon of Liverpool*, who wrote an *Account of the Extraordinary Medicinal Fluid, called Aether,* published in 1761, both as an "endeavour to extend its Utility" and also as a good opportunity to advertise the fact that he was offering "Aether" for sale, "in its Perfection", at 2*s.* the ounce, in specially marked and distinctive phials. His inhalation method, for "stubborn" headache for instance, was "to snuff a little of the Aether up the Nostrils" or "to apply a bit of Linen Rag, wetted with Aether up the Nostrils". He considered it also as "an excellent Remedy in most nervous Diseases, particularly Fits of all sorts".

A quarter of a century later the price had gone up, for William Tickell, chemist and surgeon of Bath, was selling little bottles of his "Aethereal Anodyne" at 3*s.* 6*d.* In 1787, he published an account, loud with praise, of this patent elixir. "I am almost confident," he said, "that this medicine taken twice a day in a due dose in some pectoral or mucilaginous draught, and likewise received into the lungs by inspiration, by means of Dr. Mudge's inhaler, will do more service in diseases of the lungs, than the whole farrago of Syrups, Oils, Emulsions and Conserves; or the whole tribe of acrid and indissoluble Gums, or fragrant and costly Balsams." His 'Anodyne" had many other virtues besides and Tickell felt that it was no more than his plain duty to make these known, but he also admitted that he had his "own private emolument in view" as well. He included in the second edition of his pamphlet a long and violent attack against another physician, J. M. Adair (1787) (formerly known as James Makittrick), who had had the impertinence to speak of Tickell's "quack aether" and to say that its method of preparation "savours more of empirical trick than of chemical skill" and that the product was inferior to "the aether of the shops".

There was much bad feeling on both sides and the impression is that the quack was calling the charlatan black.

Ether had been advised by Tickell for relieving, amongst other things, disorders of the stomach. James Lind (whose use of ether to cure the cough of "an officer of the excise" in 1797 has already

* It was through attending Turner's lectures at Warrington Academy, that Priestley was first introduced to the subject of chemistry.

been mentioned in a previous article) (Slatter, 1960a) described in 1785 five cases in which "gout of the stomach" had been instantly removed by ether, from which success he was "inclined to believe it a pretty certain, if not infallible remedy for that purpose". Other miscellaneous uses to which ether was put at about this time include drinking it as a diuretic and to dissolve biliary calculi, and applying it externally to reduce hernia and remove rheumatism.

As one might expect, the use of ether in medicine reached a peak in the late eighteenth century, in the period of pneumatic medicine (Slatter, 1960b). Ether was then extensively inhaled in the treatment of phthisis (consumption) "after the manner recommended by Dr. Pearson of Birmingham" (1769 a, b). This "practitioner of eminence" wrote, that he had used "vitriolic aether" for the last two years for this disease, and had "experienced the best effects" from it. It was not, however, to "supersede the use of other medicines, but to be employed in conjunction with them"; ultimately the fever and inflammation would be removed and the lungs cleansed and healed. It would be "adopting the language of quacks and insulting the understanding of everyone experienced in the profession" to assert that "beneficial consequences" would result in every case; nevertheless Pearson insisted that experience had confirmed that some "good effects" were obtained "in most instances". For children, the method was to wet a handkerchief with ether and to hold it to the nostrils; for adults, a teaspoonful or two of pure ether, or ether impregnated with powdered cicuta (hemlock), was inhaled from a teacup or wineglass. To prevent waste, it was a good idea to invert a funnel over the cup and to inhale through the tube; in winter "evaporation may be promoted" by standing the cup in a basin of hot water. Pearson (1795) wrote a circular letter to his medical friends about this new cure of consumption, so that it might be given a fair trial by other physicians and, for some years, it enjoyed great popularity.

William Withering, another eminent physician of Birmingham described, in 1798, a convenient method of inhaling ether or other volatile substances. This was by means of "a glass vessel resembling what the chemists call a tubulated receiver" (fig. 1). Air entered the globe through

FIG. 1
Withering's apparatus, 1798.

the conical tube and, mixing with the substance contained in it, was drawn into the lungs by applying the mouth to the cylindrical neck. The globe, only 3½ inches in diameter, was warmed by being held in the hand, but it could be rested on a cup of hot water if necessary. This apparatus has a special interest for anaesthetists as it will be recalled that Morton took just such a vessel—a tubulated receiver—to make his inhaler for that famous demonstration of ether anaesthesia in October 1846.

A domestic version of Withering's apparatus was "the common tea-pot" with ether inside and the spout used as a breathing tube. One advocate of this method was Dr. Thornton, physician and botanist, who made "the administration of factitious airs a leading feature of his practice", but he "paraded" them before the public in such a manner as to cause "no little dissatisfaction" amongst "the more respectable of his brethren". Thornton (1795) injudiciously advised holding the teapot "near a candle", keeping the thumb on the spout. "When the vapour begins to press upon the thumb", the teapot spout was to be put into the mouth and "the air drawn into the lungs". As long as this was done smartly presumably no harm would come from the procedure, but, immediately after describing this method, Thornton went on to speak of his experiments on the explosiveness of ether in which he got a bang "nearly as loud as a gun" and which might well have been the experience of the patient also. In general it would be far safer to follow the advice, that the inhaling of ether (or of any other inflammable gas) "should be conducted at a distance

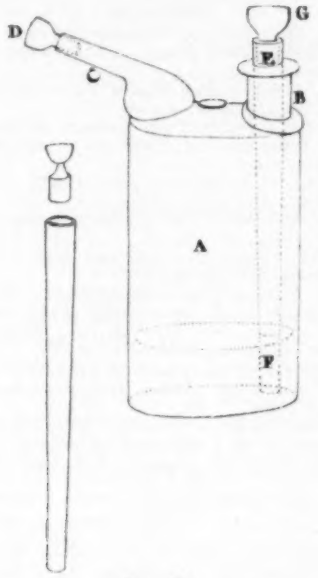

FIG. 2
Boudet's apparatus, 1811.

from a candle, lest the gas should catch fire, and at least occasion a surprise"!†

Another apparatus of interest was "Boudet's bottle" (1811). It had no expiratory valve but otherwise was similar to Magellan's modification of the Mudge inhaler (Slatter, 1960a) which was well known in France; both closely resemble the popular form of many anaesthetic inhalers. The French pharmacist, J. P. Boudet, designed it for "the inspiration of different ethers", a "widespread" practice at that time. The "bottle" (fig. 2) consisted simply of a two-necked jar of approximately 250 ml capacity. A long tube (EF), 10–12 mm in diameter, was inserted through the short neck (B) and dipped into the ether which was inhaled through a mouthpiece (D) applied to the end of the bent tube (C).

† "If you place a lighted candle to a bottle containing Ether, it will explode, and an instance is related of a French chemist who had his house blown up by this means" (Brookes, W. P. *Practical Remarks on . . . Ether*, London, 1847). In the early days of anaesthesia several such warnings were published and surgeons were urged never to perform ether operations by candlelight.

ETHER FROLICS

Although ether was still frequently used in the 1820s, it was losing its popularity as a medicine and was being inhaled mainly as an intoxicant. It had been found to have very similar properties in this respect to nitrous oxide (laughing gas), and both of them were often inhaled for fun. A current report comments that patients to whom ether had been prescribed for the relief of various minor ailments, such as neuralgia and headache, "have fallen into the habit of taking it like spirits, in the belief that it was a less immoral and vulgar method of getting intoxicated than by means of common spirits. Ladies especially have been known to get into the habit of taking ether to this extent"; it also can cause addiction.

One example takes us right back to the days of Beddoes and the Pneumatic Institution at Bristol (vide Slatter, 1960b). An "eccentric practitioner" living there was accustomed to inhale an ounce or two of ether several times a day, sitting in a drug shop and sniffing the vapour "with manifest placidity and self-enjoyment." Its effect on him he expressed as "Soothing, soothing . . . to an immeasurable degree". Not all were so quiet, however. Under the influence of both ether and nitrous oxide, some people would fling themselves about with great abandon, much to the amusement of the onlookers and the apparent enjoyment of themselves. Such "frolics" were particularly popular in America in the early 1840s and it was there that there was first appreciated the significance of the fact that cuts and bruises were sustained painlessly. This led directly to the discovery of anaesthesia.

In the early days of anaesthesia, many doctors recollected experiences of "frolics". Inhaling ether was considered "a great source of fun" amongst medical students and one doctor even had "a distinct remembrance of two cases of insensibility being produced by the inhalation of ether from a bladder".

On the other hand it is clear from the remarks made in the medical literature of 1846–47 that ether was by then no longer being used as a therapeutic agent, since such applications were especially quoted as matters of great interest.

Ether has in fact passed through waves of popularity, being used more exclusively for one purpose at one time and for another purpose at

another time. Even for anaesthesia, ether, after a year of successful employment, was almost completely dropped in Britain for nearly thirty years in favour of the far less dependable chloroform, introduced as an anaesthetic in the autumn of 1847. "For the first six months of the present year (1847), the medical and political journals were filled with accounts of the most marvelous results obtained by the administration of the vapour", but "for some months past, ether-cases and ether-vapour apparatus have been at a discount . . . Sic transit gloria". It was not until 1872, when it was yet again introduced into England from Boston, America, that ether became re-established as the anaesthetic of choice for general surgery. Since that time, ether has maintained its place as an anaesthetic agent of reliability and efficiency.

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THE EVOLUTION OF ANAESTHESIA

2. THE FIRST ENGLISH ETHER INHALERS*

BY

ENID M. SLATTER

Nuffield Department of Anaesthetics, University of Oxford, England

ETHER anaesthesia was discovered in America just over a century ago and was first publicly demonstrated on October 16, 1846, by W. T. G. Morton at the Massachusetts General Hospital, Boston. The news of it spread rapidly and two months later it had begun its triumphal career in England. But since the deliberate administration of ether vapour by inhalation to the extent of producing insensibility was an entirely new therapeutic measure, special apparatus for the purpose had to be invented. Doctors and instrument-makers wasted no time in devising, from what they already had, something that would answer the supposed requirements. At first there was a spate of ether inhalers, but experience soon revealed drawbacks in them and, before long, they had given place to new models and different techniques. In fact, fashions in ether apparatus changed very rapidly in the first few months of 1847 and any fresh idea would start a new vogue; before the year was half over, however, all these inhalers had been discarded and anaesthesia was brought about simply by means of an ether-soaked sponge. By the end of the year, ether itself had been abandoned in Great Britain superseded by chloroform, and it was not in general use again until nearly thirty years later.

In spite of their very short life, these ether inhalers have an interest all their own, particularly the very earliest ones which were adapted from existing apparatus. Three of these have their origins in the late eighteenth century, a period when much pioneer work was done on gases and inhalation, work that was to lead to many things, not least of which was the discovery of anaesthesia itself.

*The theme of these articles was taken from the Clover Lecture delivered by Sir Robert Macintosh at the Royal College of Surgeons in March 1952.

THE ETHER INHALERS OF HOOPER AND SQUIRE, MADE FROM NOOTH'S APPARATUS

I

"The year 1846 seems in a fair way to be known as the *Annus Mirabilis* of Surgery. The profession and the public in both hemispheres are in a complete ferment, consequent on the discovery by Morton." The news of this discovery—the inhalation of ether to prevent the pain of operations—was sent to England by Dr. Bigelow, Professor of the Medical School of Harvard College, Boston. On November 28, 1846, he wrote to his friend Francis Boott, a physician in London, of the discovery and also enclosed a report on the subject by his son, H. J. Bigelow. Boott (1847), realizing the extreme importance of this information, wrote immediately to the *Lancet*, enclosing the letter and report, so that the medical world should be informed of it as quickly as possible. Boott was naturally very keen to try the experiment for himself and, before his first communication to the *Lancet* was published, he had sent to the editor short accounts of the first ether operations in England: some dental extractions had been made at his home on December 19 and, two days later, the great surgeon Robert Liston, whom he had informed privately of the American discovery, had performed two major operations under the influence of ether at University College Hospital.

The inhalers of Hooper and Squire, December 1846. Boott, with a surgeon-dentist Mr. Robinson, had discussed the construction of a suitable inhaling device with a chemist, Mr. Hooper of Pall Mall; Liston had also been to see a chemist, Mr. Peter Squire of Oxford Street, who made an apparatus for him and conducted the inhaling process. Both these inhalers, which look remarkably alike, had been quickly contrived from

materials ready to hand, and in the descriptions the name of the glass vessel used was disclosed. It was "the bottom part of a Nooth's Apparatus", but it was said that "a common Mudge's inhaler" —a therapeutic steam inhaler—would have done equally well. The vessels contained ether-soaked sponges and the vapour was drawn into the lungs through a long flexible breathing-tube. These inhalers were subsequently modified and the improved models (figs. 1 and 2) were often used for giving ether in the first months of 1847.

II

The source of these anaesthetic inhalers was the apparatus by Dr. John Mervin Nooth, originally designed for making mineral waters. He had made it in order to replace the more primitive arrangement of Joseph Priestley who, in 1772, at the request of the Admiralty, had drawn up his *Directions for Impregnating Water with Fixed Air* for the use of the Navy. This artificial Pyrmont water would not only taste well, but the fixed air (carbon dioxide) in it would give the

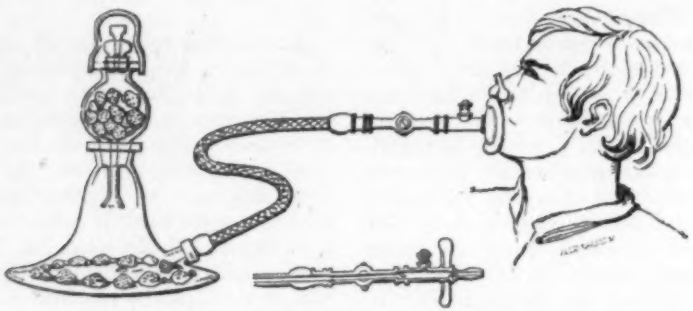

FIG. 1
Hooper's Ether Inhaler, 1846.

FIG. 2
Squire's Ether Inhaler, 1846

water an added "briskness" and, he hoped, would also help to prevent or cure sea-scurvy. The antiputrescent qualities of the gas had recently been determined and its use in putrid disorders, such as the sea-scurvy, proposed. In his pamphlet, Priestley gave some "hints" for possible uses of fixed air in medicine and said that some of his medical friends were already trying out his ideas; in his collected works *Experiments and Observations on Different Kinds of Air*, 1774–77, he gave fuller accounts of them. He suggested the inhalation of the gas for ulcerated sore throat and lung diseases; the application externally of the gas to the sanies of cancers and internally, in the form of a clyster (enema), for cases of putrid fever. These were the beginnings from which pneumatic medicine grew (Slatter, 1960).

Priestley's method (1772). The "simple, but ingenious contrivance of Dr. Priestley" for making Pyrmont water is shown in figure 3. (The phial (*e*) contained chalk covered by water. When

oil of vitriol (sulphuric acid) was added, fixed air (carbon dioxide) was generated and collected in the bladder (*d*). This was pressed to expel the gas along pipe (*c*) and into the large glass jar (*a*) which had been filled with the water to be impregnated and stood inverted in a basin of the same water. The carbon dioxide at first took the place of the water in the jar, but when the jar and basin were shaken gently, the water absorbed the gas and again rose into the jar. This process was repeated (the phial being occasionally shaken to generate more gas) and the water was ready when no more gas could be absorbed by it. If not used at once, the Pyrmont water was to be kept in a well-corked bottle. Priestley added several "observations" on the process including the fact that "if the use of a bladder be objected to, though nothing can be more inoffensive" it could be omitted, particularly if a small phial (fig. 4, a) was interposed between the larger phial containing the chalk and water and the glass jar. Then when the large phial was shaken any matter thrown up the tube (b) would collect in the small phial and not pass via tube (c) into the water to be impregnated.

FIG. 3
Priestley's Pyrmont Water Apparatus, 1772.

FIG. 4
Priestley's Pyrmont Water Apparatus, 1772.

III

The "elegant and well contrived apparatus" of Dr. Nooth was designed because of certain objections to Priestley's method. Nooth said that not only was considerable "address" required in the operation of Priestley's method, but that there was "another objection"—the bladder "was thought to render the water offensive". He had made some experiments with it and said: "It always happened, that the water acquired an urinous flavour" and could not be drunk "without some degree of reluctance". He had therefore contrived his glass apparatus "keeping convenience and cleanliness constantly in view". Priestley was quick to defend his own method and wrote that he could not "easily forgive" Nooth for his objection of offensiveness, yet in his original pamphlet he had allowed this criticism. He quibbled that the words used by Nooth implied that the bad taste was not always acquired and was therefore "an accidental thing". He himself had given his Pyrmont water to "many persons of the most delicate taste, and particularly [to] many ladies", but never had there been such a complaint. Priestley also took Nooth up on several other points, but in the end admitted that he usually used Nooth's apparatus with Parker's improvements and gave an illustration and description of it.

Nooth's apparatus (1775). The original apparatus (fig. 5) consisted of three vessels, one above the other, fitting accurately together. The lower vessel contained chalk and diluted sulphuric acid to generate the carbon dioxide. The middle vessel contained the water to be impregnated. The gas

passed upwards through a special valve, filling the middle vessel and forcing the water into the upper vessel. These were then removed as a unit and shaken. The water absorbed the gas and descended again into the middle vessel. This process was to be repeated three or four times to get a strongly impregnated water. Nooth had been using this apparatus constantly for about twelve months and saw "no reason to wish for the least alteration" to it.

FIG. 5
Nooth's Apparatus, 1775 with alternative valves.

IV

Shortly after the publication of Nooth's apparatus, the "glass-man" Parker (1775), who constructed it, made some important additions and alterations. The curved neck of the upper vessel was elongated to prevent overflow and the orifice was closed by a perforated stopper. Side ports were added to the lower and middle vessels so that the acid could be poured in, and the mineral water poured off, more easily (fig. 6: compare with figs. 1 and 2). Priestley's friend, J. H. de Magellan (1777a, b), a Portuguese monk then living in London, who devoted much of his leisure to scientific pursuits, further improved the apparatus by putting a glass cock in place of the stopper in the middle vessel. He also had made a duplicate set of the middle and upper vessels with a special stand; then, when one set was taken off to be shaken, the second set could be put in its place to receive the constantly generated carbon dioxide. Thus a double quantity of water could be made without waste of time. Magellan gives precise directions for making mineral waters which, by the addition of certain salts, could be transformed into the specific waters of Spa or Pyrmont, etc.

In previous years Magellan had "sent abroad a great many copies" of Priestley's pamphlet to different parts of Europe and now reported that "a very great number, above a thousand" of Nooth's improved apparatus had been "sent off to different parts, even to the East Indies"; the apparatus is certainly mentioned frequently in the medical literature of that period. Besides the uses already mentioned, the water was found to dissolve renal calculi—of great importance, since in the eighteenth century there were many who suffered with "the stone". The apparatus could also be used to keep foodstuffs fresh; fruit placed in the centre vessel and "supplied with plentiful

FIG. 6
Parker's Improved Apparatus, 1775.

streams or fixed air every day" did not go mouldy and, in the country, a butcher and several housekeepers washed their meat with the water to keep it sweet over the hot summer days of 1778. At the end of the century people living in London could buy "at a moderate price" a "highly impregnated" water containing "an extraordinary quantity of carbonic acid gas". It was made by a secret process and the manufacturer was "a Mr. Schweppe"!

THE SPIRAL INHALERS OF SNOW, JEFFREYS AND LIND

I

Just one month after the introduction of ether anaesthesia into England in December 1846, John Snow (1813-58), a London physician who became famous as England's first specialist anaesthetist, informed the Westminster Medical Society* that he was getting an ether apparatus made on the plan of Jeffreys' inhaler, and a week later, on January 23, 1847, he exhibited it at the society's meeting. This inhaler was the result of serious consideration on the new subject of ether anaesthesia. He had not hurried to put an apparatus before the public, nor joined in the vulgar rush that followed the first ether operations on December 19 and 21 in London, but had wisely held back, observing and experimenting, for he realized that inhalation anaesthesia must be based on the physiological principles of respiration and the production of ether vapour on the physical chemistry of the agent. It was his careful work which raised anaesthesia from near quackery to the science that it is today.

He demonstrated that the strength of ether vapour varies considerably with only small differences in temperature, which "circumstance would explain, in some measure, the variety of the results, and account for some of the failures" of the many ether operations already recorded. Snow observed that "the apparatus at first used in America and in this country for the inhalation of ether were glass vessels, and generally contained sponges". They had no means of regulating the proportions of ether vapour and air and "they had a worse fault—that of being cooled by the evaporation of the ether taking place within. The air was cooled in passing through, and took up less and less vapour as the process went on, and sometimes when the patient was on the point of becoming insensible, no further effect was produced, or when seemingly insensible, he was roused up by the surgeon's knife". To obviate these difficulties Snow had had his inhaler made of metal and placed it in a basin of warm water. The advantages of these measures were soon appreciated and in future the majority of new inhalers were made of metal, with some provision for retaining an equable temperature.

Snow's inhaler (1847a, b). Snow had taken the spiral form of his ether apparatus from Jeffreys' steam inhaler of 1842, which he "had always considered the best inhaler for aqueous vapour. The coils of the tin volute are not so numerous as in the latter, but they are amply sufficient for so volatile a liquid as ether". The spiral of tin (fig. 7, E) hung down from the inside of the lid and almost reached the bottom of the container, dipping into the liquid ether. The container itself, also of tin, measured about 2 inches deep and 4–5 inches in diameter. Around the outside was coiled a tube (AB) of flexible white metal, about 18 inches long and ½ inch in diameter, through which the air travelled before entering the container. The whole inhaler was placed in water of the required temperature, usually between 60–70°F. "When the patient began to inhale, the air would gain the desired temperature in passing through the metal pipe; it would then come upon the surface of the ether, where it would have to wind round three or four times before entering the tube going to the mouth-piece, thus ensuring its full saturation, and preserving it at the desired temperature." The only valves were in the mouthpiece (D) to prevent the return of expired air into the apparatus. They were balls of light cedarwood. During the following months Snow made several modifications to this inhaler, but its basic arrangement remained the same throughout.

II

The steam inhaler referred to by Snow was one of the many inventions of Julius Jeffreys, "late in the Medical Department of India", etc. Jeffreys "yielded to a fondness for mechanics and chemical physics, and joined a pursuit of them to

*Later amalgamated with the Medical Society of London. Snow became President in 1855.

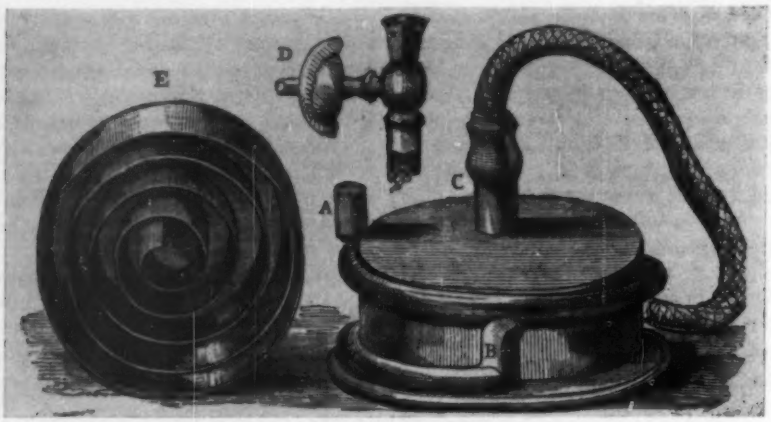

FIG. 7
Snow's Ether Inhaler (Model I), January 1847.
A—Opening of pipe at which the air enters. B—Termination of pipe in the tin box. C—Point at which flexible tube is removable by unscrewing. D—Mouth-piece. E—Tin vessel, with bottom removed, to show its interior.

the early study of the profession". This led him to seek an appointment in India, where he was better able to continue his pursuits and where he introduced various improvements into industry and agriculture. More particularly the effect of the climate upon health claimed his attention, resulting in his design for ventilating equipment and his proposal for establishing, at Simla and other suitable places, hill stations as health resorts. On returning to England in 1835, he continued his enquiries upon warmth and ventilation, considering the use of "artificial climates" in the treatment of pulmonary diseases. In 1836, he invented his "Respirator" or breath-warmer to help an ailing sister. (This prophylactic instrument became very popular and was used extensively throughout the Victorian era and on into the present century.) At the same time he designed his inhaler, but had "not thought it worth while to trouble the profession . . . with an account of it" before 1842.

Jeffreys' inhaler (1842). The beneficial effects of inhaling warm moist air to relieve a dry cough and tight chest will be readily acknowledged and could be achieved simply by breathing the steam arising from a basin of hot water. But there were also several domestic inhalers to be bought, and Jeffreys cited those of Drs. Mudge and Ramadge. However, Jeffreys found that there were certain disadvantages in these inhalers and so he designed one of his own. In his apparatus (fig. 8), the air, entering at A, passed over the surface of hot water (C) through a spiral passage (E), and so became thoroughly warmed and saturated with moisture, yet could be drawn with perfect ease into the lungs through tube (D).

Jeffreys does not give any information about the origin of his inhaler, but later in his article he mentions Dr. Beddoes' (1801) work in the same field—that of treating pulmonary invalids by artificial climates. His knowledge of Beddoes' writings was apparently scanty. He may, therefore, not have read Beddoes' other publications on the use of "factitious airs", in which case he would have been ignorant of an inhaler, very similar to his own, described some forty years earlier. It was devised by Dr. Lind who took his idea from Watt's refrigeratory.

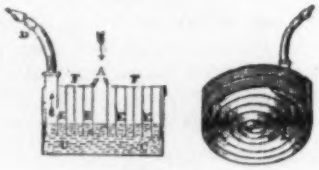

FIG. 8
Jeffreys' Spiral Inhaler, 1842.

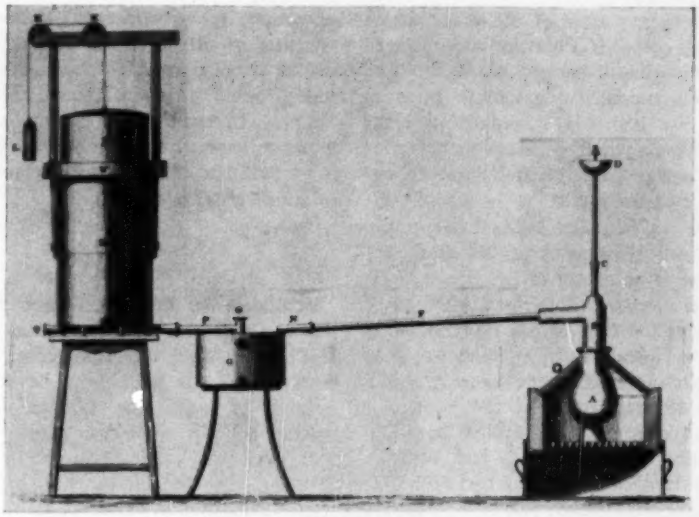

FIG. 9
Watt's Apparatus for Procuring Various Kinds of Air, 1794.

III

Thomas Beddoes had enlisted the services of the celebrated engineer James Watt to design the special equipment for his laboratory. There, both known gases and curious airs of Watt's own devising were produced, to be breathed by Beddoes' patients and later by those attending at the Pneumatic Institution.

Watt's refrigeratory (1794). The gas, generated in a cast iron "alembic, or pot" (fig. 9, A) heated in a furnace, passed down a tube (F), through the refrigeratory (G) to be cooled and on into a hydraulic bellows (H), where it was collected. An ordinary refrigeratory in distillation consists of a vessel of cold water containing a worm, or coiled pipe, through which the vapour passes and is cooled. In Watt's refrigeratory, however, the gas actually passed directly over the water "depositing its heat, or other matters capable of uniting with water, as it passes in contact with it". It was formed of a vessel (fig. 10, G) about 12 inches in diameter and 7 inches deep. Into this fitted a convex cover (S), on the underside of which was soldered an open spiral of metal about 1½ inches deep. The receiving pipe (N) from the alembic was attached at the circumference, and the discharging pipe (fig. 9, O) at the centre, of the spiral. This cover was fitted inside the vessel which was nearly filled with cold water. The gas from the alembic would "glide along the spiral channel to

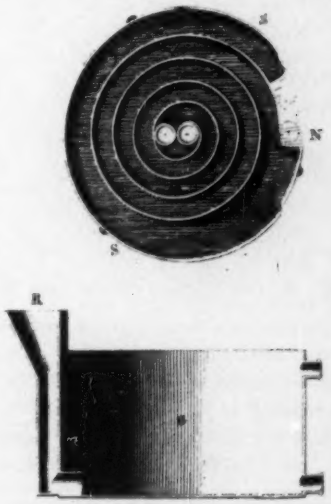

FIG. 10
Watt's Refrigeratory, 1794.

the centre", displacing some of the water which both washed and cooled it. Provision was made to change the water when it became too warm. The refrigeratory was modified for soluble gases so that they did not come into contact with the water. Watt also designed a "simplified" and a "portable" apparatus for the manufacture of airs on a small scale in the home.

Lind's inhaler (1798). Dr. James Lind, physician to the Royal Household at Windsor, described "the case of an officer of the Excise, who, during the severe weather of January, 1797, being much exposed to the cold in the exercise of his duty, had got a violent cough", which was "followed by the symptoms of a rapid consumption". The remedies for this included the inhalation, several times a day, of ether vapour in which powdered cicuta (hemlock) leaves had been infused. In a few days "almost all the bad symptoms were wonderfully diminished; yet, finding that he got hardly any sleep at night, and that he had been a bad sleeper for above a year", Lind "made him inhale about a quart of hydrocarbonate gas, diluted with about fourteen quarts of common air".* The officer enjoyed "an uninterrupted night's rest" and, by continuing the inhalations, was able to return to duty in about a month.

Lind added a special note that the gas was drawn in "over the steam of hot water" in an inhaler "constructed on the plan of Mr. Watt's refrigeratory". In a footnote it was explained that "this inhaler is made of japanned tin, and being filled with hot water, is interposed between the bag or receiver of air and the mouth of the patient, so that the air is heated by the vapour of hot water in passing through it". Unfortunately no illustration was given, but, excepting that the air was artificial and came from an air-bag, whereas Jeffreys' patient breathed free atmospheric air, the two inhalers cannot have been very different.

DR. MUDGE'S INHALER

The first notice on ether anaesthesia appears in the *Pharmaceutical Journal* of January 1st, 1847, where there is a sketch of the inhaler Squire made from a Nooth's apparatus for the first major operations to be performed under the influence of ether in this country. The editor makes the useful suggestion that "a common Mudge's inhaler" could be used instead. This was a very simple therapeutic appliance for inspiring aqueous vapour, contemporary with the Nooth's apparatus and, like it, easily convertible for the administration of ether.

I

John Mudge, the inventor of this popular "inhaler", was the son of the Rev. Zachariah Mudge and was born at Bideford, Devon, in 1721. Although "not altogether with the full approval of his father" he nevertheless "determined to enter the medical profession" in which he was to attain great eminence. The family went to live in Plymouth in 1732 and there Mudge remained all his life, indifferent to the suggestion that a move to London would considerably advance him in the profession. As well as being a distinguished physician, Mudge was also a mechanic of genius and his work on reflecting telescopes earned for him the Copley Medal of the Royal Society in 1777 and lasting renown in the field of astronomy.

"He was a man of great amiability of character, genial and pleasant in his manner, idolized by his family, and beloved and respected by all who knew him. His society was much sought after", and it is recorded that one patient who was sent to him was assured "that if his physic did not cure her, his conversation would". He numbered amongst his close friends and guests many famous men of the day, including such names as Sir Joshua Reynolds, Dr. Johnson and John Smeaton. Mudge was of a "tender constitution" and in the latter years of his life he suffered much with the gout, the pain of which he bore, however, with "the greatest fortitude" (Flint, 1883). He passed peacefully away in 1793 in his seventy-second year.

Mudge's inhaler (1778). "The ingeniously contrived machine, which he called an Inhaler" was described by Mudge in his small book, *A Radical and Expeditious Cure for a Recent Catarrhous Cough*, 1778. It was made of pewter and "resembles a porter-pot" (fig. 11). The handle was hollow and had three holes (f) bored into it at the top and one hole bored through to the inside

*Hydrocarbonate gas was made by passing steam over the red hot charcoal of certain woods specified by Watt.

FIG. 11
Dr. Mudge's Inhaler, 1778.

at the base. At the side of the lid was a chamber covered by a grating (a), which held a small cork ball-valve (b); in the centre of the lid was a short flexible breathing-tube (d) and mouth-tube (e). To use it the patient was directed to lie in bed, well covered up, and the inhaler, which held about a pint, was three parts filled with hot water, wrapped in a napkin and placed in his armpit. He breathed in and out through the inhaler for 20 minutes to half an hour without taking the tube from his mouth. The inspired air entered by the hollow handle and bubbled up through the water, becoming both warm and moist; the expired air passed over the surface of the water and escaped through the valve. Both the top of the handle and the valve-box had of course to be kept free of the bed-clothes.

Mudge claimed for the inhaler two distinct advantages which gave it "superiority to all others". Firstly, the fact that the tube was retained in the mouth throughout the inhaling period, so that children and others who "for want of skill in the use of the common sort, arriving from the necessary interruption in breathing" were no longer deprived of the benefit of the treatment. Secondly, the warm and moist expirations being caught under the bed-clothes, produced a sweat which both relieved the dryness of the skin and also provided "the most eligible evacuation for removing the fever". If this inhaler was used the very same evening of the attack, the patient was "sure of being surprised with an immediate cure". Mudge, who was himself "the first subject of the experiment", could scarcely believe it and said, "It was with difficulty and not till after several trials, that I could myself credit the reality of the cure". If the cough was several days old, the inhaler was to be used both at night and in the morning. Mudge cautioned anyone from making a trial of the inhaler with cold water inside "as it will certainly produce the complaint for which it is . . . proposed as a remedy". He added another warning in a later edition of his book—that he had known "several people of delicate constitutions, and tender lungs, get severe colds by incautiously reading a damp news-paper"!

II

In the same year that Mudge's book was published, J. H. de Magellan (1778), always quick to make new scientific discoveries known abroad (as

we already know in the case of the mineral-water apparatuses), wrote in a most enthusiastic manner to a medical friend and compatriot in Paris, giving him details of this infallible cure. He repeated the salient points from Mudge's account and added a drawing of the inhaler, which he preferred to call "Respirateur" (fig. 12). Although Magellan had made a slight modification to it, the principle remained the same. Instead of by way of a hollow handle, the air entered down a long tube (cd) which passed through the lid into the water. It was covered, like the valve-box, by a perforated cap.

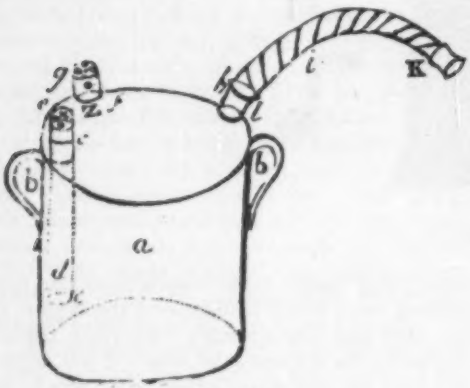

FIG. 12
Magellan's Apparatus, 1778.

The inhaler soon passed into general use and is frequently mentioned in medical literature from that time. It was also given a place in the many encyclopaedias of the early nineteenth century. But although the idea was good, the inhaler was found not to work well in practice. Dr. Withering (1798), well known as both physician and botanist, made the good point that if all "the joints of the machine" were carefully closed "so as to guard against deception" and to force all the inspired air to pass through the body of water, so much resistance was caused that breathing was made "very laborious and troublesome even to persons in health and strength, and utterly impracticable to invalids". He should have added that easy breathing would be impossible anyway, with such small air holes and very narrow inhaling-tube. Many of the early ether inhalers had been based on steam inhalers such as Mudge's, and many difficulties had been encountered because of these resistances, but after the first few weeks of anaesthesia, ether inhalers were made with special wide breathing-tubes and enlarged apertures.

Although in 1818 Dr. Thomas Machell, introducing a somewhat similar inhaler of his own, remarked that the apparatus with which "the late Dr. Mudge obliged the world" had "fallen into disuse", since its management in other hands "was attended with insuperable difficulties"; yet later, in 1829, another physician, Sir James Murray, this time boosting iodine inhalation (in a book *A Dissertation on the Influence of Heat and Humidity* which earned for him his knighthood and the position of resident physician to the lord-lieutenant of Ireland), said that "most hospitals are furnished with inhalers like Dr. Mudge's", which he disparagingly called a "tin tea-pot".

III

The spiral inhaler of Julius Jeffreys which Snow later adapted for ether anaesthesia, was originally made to replace the existing steam inhalers, which Jeffreys (1842) had found to be unsatisfactory. These, he said, were of three kinds. In the first kind, such as "Dr. Mudge's inhaler, when acting according to its principles" and one by Dr. Ramadge (a specialist on chest diseases), the air, rising up through the body of the water, was made too hot at first but cooled rapidly; there was also the above-mentioned objection of the difficulty of inhaling against the pressure of water. In the second, "Dr. Mudge's inhaler as commonly made", the air passed only over the surface of the water and, although this was "the preferable plan", in so short a transit the air scarcely had time to become sufficiently warmed and moistened. The third type of inhaler referred to by Jeffreys was a metal tube which "winds like the worm of a still through hot water". This warmed, but did not moisten, the air, which Jeffreys thought "ill suited . . . for almost any conditions of the lungs". He was probably thinking of another apparatus by Dr. Ramadge who was, however, following a different argument—that weak lungs should be strengthened by forced respiration and that the production of asthma or cough will arrest the development of consumption.

IV

Jeffreys was writing in 1842 and it is clear that the Mudge inhaler was still the most popular one for administering aqueous vapour, so it is no surprise that, in 1846, this was the apparatus suggested, to those unable to procure the new device of Squire, as being suitable for ether anaesthesia. From about March 1847, Mudge's word "inhaler" was, for the first time, generally substituted for the lengthy term, "apparatus for inhaling the vapour of ether". By 1846, it was already nearly seventy years since the Mudge inhaler had first made its appearance and it was interesting to see that it was still being advertised in instrument catalogues at the end of the nineteenth century. It was, however, a surprise to find a modern advertisement of the inhaler only a few years ago and to have an order for it complied with at once. The Mudge inhaler supplied looks just like the original picture, except that the tubing is modern (though still narrow) and the handle no longer bears the delicate leaf moulding.

Although it has not been used for the relief of catarrhous cough, the inhaler has been breathed through and the objection of pressure resistance was found to be quite true. Also the cork ball-valve in this apparatus was found to stick, so that the expired air either raised the lid off the inhaler or bubbled back up the handle. Either way, unless the user breathed out very gently, water flooded out liberally, a rather unpleasant occurrence for a patient lying in bed! Nevertheless, in spite of these troubles, the fact remains that the inhaler is made still, now nearly two hundred years since its origin—a lasting memorial to its celebrated inventor, John Mudge.

ACKNOWLEDGMENT

I should like to thank members of the Nuffield Department of Anaesthetics, especially Dr. H. G. Epstein, for the help they have given me.

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INDUCTION OF ANAESTHESIA IN THE FOOT-DOWN POSITION

Sir,—With reference to the article of Drs. Snow and Nunn on induction of anaesthesia in the foot-down position (*Brit. J. Anaesth.*, 1959, **31**, 493), you might consider the following two cases worthy of publication.

Since the suggestion of Morton and Wylie (1951) it has been my practice to induce in this position all cases of intestinal obstruction, emergency Caesarean section and any others in which a full stomach is likely. I have not the figures available but the total cases number many hundreds.

Case 1. Laparotomy for acute intestinal obstruction in a female aged 75 years. A Ryle's tube had been inserted in the ward and the stomach contents aspirated previous to the induction of anaesthesia. Following premedication with pethidine 50 mg and atropine 0.6 mg anaesthesia was induced with thiopentone 300 mg in 2.5 per cent solution mixed with gallamine 80 mg.

As the laryngoscope was being passed into the pharynx the patient's left arm slipped off the table and a nurse standing on the other side reached across to collect it, pressing on the abdomen in the process. I watched a tide of brown liquid rise out of the oesophagus into the pharynx. On instructing the nurse to remove the pressure on the abdomen, the tide receded without entering the larynx and the trachea was intubated. Result—full recovery without complications.

Case 2. Laparotomy for acute abdomen (undiagnosed) in a male aged 60 years. This patient had been vomiting copiously and would not tolerate a Ryle's tube. Following premedication with pethidine 50 mg and atropine 0.6 mg, anaesthesia was induced with thiopentone 500 mg in 2.5 per cent solution mixed with gallamine 100 mg. This dose of gallamine was not enough to produce complete apnoea and as laryngoscopy was being performed two spasmodic contractions of the diaphragm occurred. This produced a gush of fluid stomach contents up the hill into the pharynx, which was controlled by suction whilst the trachea was intubated and the cuff inflated. A wide-bore stomach tube was then passed and 4 pints of brown fluid aspirated. A further half-pint was expressed from the dilated stomach by the surgeon when the abdomen had been opened. The cause of the condition was found to be a mass of adhesions kinking the upper end of the jejunum. The patient recovered without postoperative complications or any signs and symptoms of inhaled stomach contents.

Without the foot-down position, both these cases could have ended in fatal asphyxia.

DONALD BLATCHLEY
Chiswick, London

Sir,—In the issue of *B.J.A.* (1959, **31**, 464) Drs. Parkhouse and Simpson in "A Restatemen. of of Anaesthetic Principles" say of the Ayre's T-piece:

> " . . . it has proved to have mathematical and physical characteristics which defy all attempts at calculation. Despite many variations of the T-piece, speculation still runs high as to what flowrate of gases is required relative to any particular length of tubing, in order to ensure adequate elimination of carbon dioxide."

In fact a fairly detailed analysis of the mathematical and physical characteristics of the T-piece has been made by Dr. W. W. Mapleson, Ph.D., of this department (Mapleson, 1954). The results of his analysis agree with the experimental findings of Woolmer and Lind which were made as far back as 1954.

WILLIAM W. MUSHIN
Welsh National School of Medicine, Cardiff

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